ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY.

VOL. XXII. SEPTEMBER, 1913. No. 3.

XXXV.

DISEASES OF THE LABYRINTH WITH A REPORT OF NINETEEN CASES.*

BY FRANK R. SPENCER, A. B., M. D.,

BOULDER, COLO.

A brief review of the anatomy and physiology of the semicircular canals may not be out of place before considering diseases of the labyrinth, especially since it has been only a few years that this branch of otology has been well understood. The horizontal or external semicircular canal lies almost horizontally, as its name implies. The ampulla is anterior, while the smooth end is posterior and a little lower, so that the canal is slightly oblique. The superior or frontal semicircular canal is almost vertical with its ampulla anterior and external, adjoining the ampulla of the external canal. The smooth end is, therefore, internal and posterior to the ampulla and unites with the smooth end of the posterior canal to form a common opening into the vestibule. The posterior or sagittal canal

*Presented as candidate's thesis at the nineteenth annual meeting of the American Laryngological, Rhinological and Otological Society, held at Washington, D. C., May 8-10, 1913.

is also oblique, with its ampulla opening into the lowest and most posterior portion of the vestibule. I have given the positions of the canals, because it better enables one to understand the direction of the movement of the endolymph during the subsequent tests.

According to Ewald's law, the movement of the endolymph from the smooth end toward the ampulla is the more effective movement in the external or horizontal semicircular canal, and produces nystagmus to the same side. Conversely stated, the movement of the endolymph from the ampulla toward the smooth end is the less effective movement and produces nystagmus to the opposite side. If we consider the superior or frontal semicircular canal and the posterior or sagittal, the law must be reversed.

With Ewald's law in mind, we have at least the five following methods of testing the reaction of the labyrinth: First, the turning test. Second, the caloric test. Third, the finger test. Fourth, the fistula test, and fifth, the galvanic test. The turning test has the advantage of testing both ears at the same time, while its disadvantage is that each ear cannot be tested separately. However, if one labyrinth is more irritable than the other, the more irritable one determines the direction of the nystagmus. In case one labyrinth has been destroyed by disease or by an operation, we are able, of course, to test only the other internal ear by turning. By the caloric and galvanic we may test either ear separately or the two together.

Bárány has demonstrated that turning either to the right or to the left ten times produces the best reaction.[1] If we turn a patient with normal ears to the right ten times and stop, he should have, immediately after the turning, a horizontal nystagmus to the left for fifteen to thirty seconds—that is, the quick component should be directed to the left. Conversely, after turning to the left ten times and stopping, there should be a horizontal nystagmus to the right for fifteen to thirty seconds. To test the external or horizontal semicircular canals by turning, the patient should sit upright in a straight back revolving chair with the head erect. The superior canal may be tested in a similar manner, if the patient inclines his head forward during the turning, and the posterior if he inclines his head backward, so as to have the head in either position in the horizontal plane. The last two canals

produce, of course, a rotatory nystagmus. In Prof. Urbantschitsch's clinic in Vienna (which was formerly in charge of Politzer) Bárány, Neumann, Ruttin and others use a light weight revolving arm chair with an iron rod extending from the back of the seat up through the back of the chair for a distance of one meter above the top of the back. This rod is about two and one-half centimeters in diameter and is grasped by the physician above the patient's head during the turning. It is quite important to have each of the ten turns of uniform rapidity and to stop the patient as quickly as possible at the end of the tenth turn. The patient should wear dark smoked glasses during the turning.

The caloric test may be made with either hot or cold water, if the drums are intact or if suppuration is present. If there is a perforation with an absence of pus (healed chronic purulent otitis media), hot and cold compressed air may be used in order to avoid moisture. Syringing with cold water normally gives nystagmus to the opposite side, and with hot water to the same side. Cold water is usually more agreeable to the patient and should have a temperature of about twenty-five degrees centigrade, although it may be necessary to use water of fifteen degrees in some cases in order to obtain the reaction. The hot water should have a temperature of about forty-eight degrees centigrade.

The finger test may be made after syringing the ear. After cold syringing the patient falls toward the syringed ear, if asked to stand erect with the eyes closed, and misses the finger of the physician by passing his finger to the left of the observer's if the left ear was the one syringed. The above test is the reaction of a normal ear to cold syringing, and the condition would be reversed if hot water were used.

The test for a fistula may be made by either compression or aspiration. A fistula is usually located in the bony portion of the horizontal or external semicircular canal, as this is the most exposed of all the semicircular canals to middle ear suppurations. However, it may be in the cochlea, vestibule or one of the other semicircular canals. Ruttin states that in forty out of fifty cases in which a fistula was demonstrated, compression of air in the external auditory meatus and middle ear produced nystagmus to the same side, and aspiration produced nystagmus to the opposite side. In other words, this

is the typical reaction of the labyrinth to condensation and rarification of air in the middle ear, and is the rule. This shows that the endolymph is driven by compression from the smooth end toward the ampulla, which is the more effective movement for the horizontal semicircular canal and, therefore, produces nystagmus to the same side. If we use the fistula test while the patient is under the influence of a general anesthetic on the operating table, we produce only the slow component from the labyrinth, as the quick or central component is absent during the third stage of ether or chloroform anesthesia. The author wishes to emphasize the importance of making the test for the fistula symptom, first, because it yields, when present, valuable information, and, second, it is an easy test to make. If present in any case of chronic purulent otitis media, we are almost certain to find a cholesteatoma in the middle ear and antrum as the etiologic factor in producing the erosion of the outer bony labyrinth.

The four tests just given all act on the endolymph in the labyrinth, while the galvanic test determines the reaction of the eighth nerve. If we place the cathode in the right meatus we should expect a nystagmus to the right, and if we place the anode in the right meatus we should expect a nystagmus to the left, if the auditory nerve is normal. The test does not depend upon an intact labyrinth, as it may be used soon after the labyrinth has been destroyed either by disease or by an operation. We may begin the test with four milliamperes of the galvanic current, but as much as eight to fifteen may be required before the nerve responds. If the nerve has been destroyed or is atrophic, it will, of course, not respond.

Ruttin has devised several pieces of apparatus for the above tests. The first consists of two aural specula mounted on opposite ends of a semicircular rubber headband so that the headband holds the two specula firmly in the ears. A small metal tube is fixed in each speculum so that it can be connected with a rubber tube and a jet of either hot or cold water thrown into the external auditory meatus from either one or both sides. The return flow passes out through the specula. This piece of apparatus makes it possible to syringe each ear separately with a uniform quantity of water equally divided between the two ears. Of course, if two normal ears in the same patient are syringed simultaneously with either hot or

cold water, there will not be any nystagmus produced, as the reaction of one labyrinth counteracts the reaction of the other. A similar piece of apparatus has been made so that a metal electrode is held firmly in each external auditory meatus by the rubber headband, so that the reaction of either the right or left auditory nerve may be tested without changing the apparatus.

Three degrees of spontaneous nystagmus are recognized: First, spontaneous nystagmus when the patient looks in the direction of the quick component, but which is absent when the patient looks either straight ahead or in the direction of the slow component. Second, spontaneous nystagmus is present when the patient looks straight ahead, but is increased by looking in the direction of the quick component. The nystagmus disappears, of course, when the patient looks in the direction of the slow component. Third, the third degree is present in all positions of the eyes, but is greatest when the patient looks in the direction of the quick component, and is least when the eyes are turned in the direction of the slow component. Sudden destruction of one labyrinth always produces third degree nystagmus with the quick component directed away from the diseased ear. The slow component is the peripheral one, and is due to irritation of the labyrinth, while the quick component is always central and represents nature's effort to counteract the naystagmus due to labyrinthine irritation. For instance, if we syringe the ear of an unconscious patient we produce only the slow component, as the central or quick movement of the eyes is absent. This can be well demonstrated during the third stage of chloroform or ether anesthesia by syringing the ear just prior to a mastoid operation. The eyes move in the direction of the slow component and remain in that position as long as the syringing is continued, because without the central component we cannot produce nystagmus. Inflammations and suppurations of one side produce a nystagmus to the opposite side for from three to fourteen days.

Ruttin (Ruttin's Course in Surgical Diagnosis, University of Vienna. Similar table given in Prof. Urbantschitsch's Lehrbuch der Ohrenheilkunde, fifth edition, page 456) gives the following table or classification of the different forms of labyrinthitis: First, cases of circumscribed serous labyrinth-

itis give a history of labyrinthine symptoms during the attacks. There is usually horizontal and rotatory nystagmus to the opposite side, but rarely nystagmus may be directed to the same side. Hearing is present; the fistula symptom is present; and the labyrinth reacts to the caloric test. Second, cases of diffuse serous labyrinthitis do not give any history of labyrinthine symptoms during the course of the middle ear disease. Third degree nystagmus is present with the quick component directed toward the opposite side usually, although it may be directed toward the same side in mild cases. Hearing is usually absent, but may be present. The ear usually reacts to the caloric test, but there may be a total absence of any reaction. The fistula symptom is usually absent, but is present in those cases in which the diffuse serous labyrinthitis was preceded by a circumscribed serous labyrinthitis. Third, cases of acute purulent diffuse labyrinthitis do not give any history of labyrinthine symptoms during the attack of middle ear disease. Third degree nystagmus is present with the quick component directed toward the opposite side. The hearing, the caloric reaction and the fistula symptom are all absent. Fourth, cases of chronic diffuse purulent labyrinthitis give a history of labyrinthine symptoms during the attack. Spontaneous nystagmus is often absent, but may be present. The hearing, the caloric reaction and the fistula symptom are all absent. The first two types of labyrinthitis demand only a radical mastoid operation, while the third and fourth types demand a radical operation and an operation upon the labyrinth too.

Case 1.—Mr. A., age twenty-five years, examined June 29, 1911. On the twenty-seventh day of the preceding month he had had a radical mastoid operation on the right side and made an uninterrupted recovery. For a few days prior to the examination he had had severe vertigo, and the fistula symptom was found to be present upon examination. Compression produced nystagmus to the same side, and aspiration to the opposite side. Examination of the eyes showed a spontaneous nystagmus with the quick component directed to the right when the eyes were turned to the right. The nystagmus was made stronger by moving the head, which shows that he was not a malingerer. He showed a slight nystagmus to the right after syringing, but did not fall in the direction of the slow

component to the left, as we would expect. This latter test was made ten minutes after the syringing, so it is not as valuable as if made immediately after the syringing. Examination of the patient's hearing showed the following: The patient heard a conversational voice at one-half meter with the right ear; Weber's and Rinné's tests were negative; and bone conduction was slightly shortened. The upper tone limit was below normal, and the lower tone limit was elevated. Turning the patient to the left ten times produced nystagmus to the right which lasted thirty seconds. Turning to the right ten times produced nystagmus to the left for twenty-seven seconds. Syringing the right ear with cold water produced nystagmus to the left.

Diagnosis.—Diffuse serous labyrinthitis following circumscribed serous labyrinthitis. As he had had a radical mastoid operation, he did not need any operation, but he should be watched to see if a purulent labyrinthitis develops. There are three types of serous labyrinthitis so far as the hearing is concerned: First, cases with good hearing. Second, cases with poor hearing, and third, intermediate types with only a moderate preservation of hearing.

Case 2.—Mr. B., age eighteen years. Right ear normal. The history of the left ear is as follows: Eleven years ago, in the province of Galicia, he had a Stacke radical mastoid operation on the left side. After the operation the discharge ceased. One year ago the left ear began to discharge again, but he had not had any headache, vertigo or vomiting. Examination of the left ear showed the following: He heard a conversational voice at one and one-half meters; Weber's and Rinné's tests were negative. The lower limit was elevated and the upper tone limit was below normal. By Schwabach's test the bone conduction was not shortened. The caloric reaction was present; polypi and granulations were present in the antrum. Turning to the right ten times produced nystagmus to the left for fifteen seconds; turning to the left ten times produced nystagmus to the right for thirty seconds. Upon testing for the fistula symptom the patient showed nystagmus to the same side with compression, and to the opposite side upon aspiration.

Diagnosis.—Circumscribed serous labyrinthitis of the left ear. A radical mastoid operation is indicated. The prognosis

should be guarded, as there is some danger of a purulent labyrinthitis following the radical exenteration.

Case 3.—Mr. C., age thirty-four years, gave a history of chronic purulent otitis media of the right ear for the past seven years which followed influenza. One year ago the left ear began to discharge. The patient complained of headache, vertigo, nausea and vomiting recently. Examination of the left membrana tympani revealed a dry perforation in the posterior superior quadrant leading into the antrum. Examination of the hearing showed the following: A conversational voice was heard at one-fourth meter with the right ear and at two meters with the left ear; by Weber's test the fork was heard better in the left ear; Rinné's test was negative for both ears. The lower limit was slightly above normal, but the upper limit was normal. Spontaneous nystagmus was absent. The fistula symptom was present in the left ear with nystagmus to the left by compression and to the right by aspiration. Turning to the right ten times produced nystagmus to the left for thirty seconds. Turning to the left ten times produced nystagmus to the right for thirty-one seconds, which was normal for both labyrinths. As the vertigo in this case came on only fourteen days ago, and since his hearing is as good as before, he has only a fistula without any disease of the membranous labyrinth.

Diagnosis.—Circumscribed serous labyrinthitis. In this case only a radical is indicated, although we may treat the patient for awhile, as the fistula may close without an operation. If the patient is not treated he is almost sure to have a purulent labyrinthitis.

Case 4.—A young lady gave the following history: Two months ago she had a purulent discharge from the left ear, and a few days ago she began having a discharge from the right ear. She thinks she has tuberculosis, as she has been given injections and, as she does not know what the injections are for, she has been led to believe she is getting tuberculin. From her statements it was evident that she had been taking mercury, and "606" may have been injected in place of tuberculin. She stated that her hearing failed rapidly after the injections, so this would lead one to believe she had been given "606." The hearing tests were as follows: The air conduction was shortened for both ears, but the bone conduction was excellent. She

heard reasonably well with each ear when the two and one-half meter speaking tube was used. Weber's test was lateralized in the left ear and Rinné's was positive for each ear, but more positive on the right side. The upper tone limit was below normal for each ear and the lower tone limit was elevated on each side. Third degree spontaneous nystagmus was present, which was most marked when the patient's eyes were directed to the left. Turning to the left ten times produced nystagmus to the right for fifty seconds; turning to the right ten times produced nystagmus to the left for thirty-five seconds. Cold air passed into both the right and left ear successively gave negative results. With the right ear the patient heard forks D 1 to C 4, and with the left ear forks A 1 to G 3 of the Bezold-Edelman series, when the hearing should have been perfect for forks C 2 to C 8. The patient has, therefore, only the middle range of hearing. Judging from the history, the internal ear disease must have preceded the middle ear disease. The vestibular division of the eighth nerve is negative, as it reacts well, but the cochlear division is diseased.

Diagnosis.—Chronic purulent otitis media and toxic neuritis of the cochlear division of the auditory nerve in each ear. The prognosis is better if it is due to syphilis, but in this case it may be tuberculous. Lead, quinin and the salicylates have a selective action on the cochlear nerve. The patient formerly had large doses (0.5 g.) of aspirin for a prolonged period, so that the toxic neuritis may have been due to aspirin.

Case 5.—A boy, age seven years, was admitted to the otologic department of the Vienna general hospital May 15th, with the following history: Three weeks ago he had pain in the left ear, and three days ago very severe pain in the left ear and headache, but without nausea, vomiting and vertigo. Two days before admission he had a high temperature. The left membrana tympani was red and bulging and the landmarks were obliterated. The posterior superior wall of the external auditory meatus was bulging and the mastoid process was tender upon deep pressure. The right ear was negative. Functional examination of the left ear revealed the following: He heard a conversational voice from four to five meters and a whispered voice at a distance of two meters. Weber's and Rinné's tests were negative; bone conduction was lengthened. He could not hear fork C 1, but did hear fork C 4. The turning tests were normal.

Diagnosis.—Acute purulent otitis media. A free incision was made in the left membrana tympani on the day of his admission to the hospital. The temperature before admission was 39.2° C., and after the incision it fell to normal by lysis within the next few days. We may have fever if the pus extends to the dura or lateral sinus, but we are not apt to have fever from an uncomplicated middle ear abscess with good drainage. The ninth day after his admission the temperature was 38.5° C. At this time (ninth day) a simple mastoid operation was performed. The pus came from the posterior portion of the mastoid under high pressure, so that the patient had either a perisinus or extradural abscess. The lateral sinus was exposed over a large area, and a small incision was made in the sinus wall, but no thrombus was found. After the operation the fever continued with a very slight fall until four days later, when it rose to 39.6° C. A second operation was decided upon May 30th. The internal jugular vein was ligated on the left side and a thrombus removed from the left lateral sinus. The temperature gradually dropped, reaching normal on June 4th. At this time there was free drainage through the sinus, as fluid injected into the upper portion of the internal jugular above the point of ligation passed out through the opening in the lateral sinus. On the eighth day the temperature was high again, and it was impossible to find the lower end of the upper portion of the jugular, so a bulb operation was performed. (The spinal accessory nerve is over the jugular vein in sixty per cent of all cases, and it was over the jugular in this case. The bulb was laid over the nerve from above, so that the spinal accessory is now under the jugular.) Since this last operation the recovery has been perfect. The infection in this case was due to the streptococcus pyogenes, and this organism is apt to produce a sinus thrombosis.

Case 6.—A girl, age seventeen years, was admitted to the otologic department of the Vienna general hospital the day before this examination with the following history: Eight days ago she had a severe sore throat, and two days later severe pain in the left ear, with a temperature of 37.5° C. to 38.8° C. The left membrana tympani had a small perforation in the anterior inferior quadrant. The mastoid was tender upon deep pressure, especially over the tip, and the neck was rigid. Ruttin stated that the torticollis might be due to, first, Bezold's

type of mastoiditis; second, enlarged lymph glands; third, peribulbar abscess; fourth, thrombosis of the mastoid emissary vein, and, fifth, Gleissenger's sign. If pus is present around the bulb, it is likely to pass into the deeper structures of the neck and produce torticollis: If such is the case the pus must be evacuated by going down to, but not into, the bulb. The functional tests for the left ear showed the following: Conversational voice heard at thirty centimeters; a whispered voice at five centimeters. By Weber's test the sound was lateralized in the left ear and Rinné's test was negative. The air conduction for forks C 1 and C 4 was shortened.

Ruttin stated that this patient might have a brain abscess which, if present, must be a temporal lobe abscess. If the abscess were located in the cerebellum she would have vertigo and nystagmus with a disease of the labyrinth, which she did not have. She did not have aphasia, so she probably did not have a temporal lobe abscess. In the latent stage, without any focal symptoms, there is a slow pulse; no evidence of meningitis, as the reflexes are normal, and no dermagraphia. This patient must be kept under observation for a time before a positive diagnosis can be made.

Case 7.—A young lady of about twenty years of age, was admitted July 13th with the statement that she had been totally deaf in the right ear since early childhood and that recently she had lost a great deal of hearing in the left ear.

Upon testing her hearing the right ear was found to be totally deaf and the left showed the following: The conversational voice was heard from one-fourth to one-half meter. Weber's test was lateralized to the left; Rinné's was negative, and the bone conduction was shortened. She heard forks C 1 and C 4.

The eyes showed a spontaneous nystagmus of the third degree, which was probably ocular, as she had astigmatism in each eye with convergent strabismus and amblyopia of the right. Turning to the right produced nystagmus to the left of questionable duration, as the squint and ocular nystagmus greatly interfered with the test. Turning to the left ten times produced nystagmus to the right for twenty-four seconds. The finger test was negative. The right ear did not react to the caloric test, but the left reacted normally. The galvanic test was made, beginning with four milliamperes and increasing to

fifteen. However, the ocular nystagmus interfered so much that it was impossible to tell very much if anything about the accuracy of the reaction.

Diagnosis.—Congenital defect of the eyes (retinitis pigmentosa with a history of parental intermarriage) and of the vestibular and cochlear apparatus.

Case 8.—A man, fifty-four years of age, was admitted July 14th with poor hearing. In the eye clinic a diagnosis of papillitis due to suspected brain tumor was made. He had been ill for five weeks and complained of tinnitus in the left ear, but he was not very intelligent, so he could not tell what symptoms he had.

The hearing tests showed the following: He heard a conversational voice at a distance of seven meters with the right ear and at a distance of five with the left; the whispered voice at five meters with the right and at two with the left. Weber's test was not lateralized (Weber's in the head) and Rinné's was positive for each ear. The bone conduction was normal for each ear, and he heard forks C 1 and C 4 with each ear. The cochlear apparatus may be slightly affected on the left side.

Spontaneous nystagmus was absent, but the following reactions were obtained from the labyrinth: Turning to the left ten times produced nystagmus to the right for twenty-four seconds, and turning to the right ten times produced nystagmus to the left for twenty-five seconds, so both tests were normal. With the finger test, after turning to the right ten times, the patient's finger deviated to the right, and after turning to the left ten times, the patient's finger deviated to the left. This test also produced a normal reaction. For the caloric test both ears were first syringed simultaneously with cold water. For this test both ear drums must, of course, be present. There was an absence of any reaction and the test was negative. Syringing the right and left ears separately with cold water gave a normal reaction, except that the left ear was a little more irritable than the right. However, the reaction was typical, normal or physiologic.

A brain tumor in the middle fossa is not apt to affect the hearing unless the tumor is large. If it is in the posterior fossa it is very apt to affect the hearing and the vestibular apparatus very early. Tumors of the cerebellopontine angle give rise to symptoms early from the cochlear and vestibular ap-

paratus. All tumors of the brain produce shortened bone conduction just as soon as they are large enough to give rise to increased pressure. The membrana tympani may be slightly retracted, thus indicating increased bone conduction, but we find shortened bone conduction if there is increased intracranial pressure.

As this man has not spontaneous nystagmus he cannot have a brain tumor of any size, especially in the posterior fossa. If we find nystagmus due to a severe labyrinthine disease, the nystagmus disappears in from three to fourteen days. If the nystagmus is due to a cerebellar tumor, it occurs in attacks, and it may occur to either side with great irregularity. For instance, the direction of the quick component may change from the right to the left, or vice versa, while we are making the examination. However, the nystagmus is always present if the tumor is of any size. This patient cannot have a cerebellar tumor, as the reaction of the labyrinth is normal, unless the tumor is too incipient in its development to produce symptoms at this time. We must have this case return in two months for reexamination before we can be absolutely certain of the diagnosis.

Case 9.—A man in the employ of one of the steam railroads fell from the top of a box car and struck on his head. He was unconscious for fully three minutes and had a hemorrhage from both ears, but he did not have either tinnitus or deafness. The blood may come from any one of the following: First, the external meatus; second, membrana tympani; third, the middle ear; and, fourth, the labyrinth. As he had neither vertigo nor deafness, he did not have a fracture of the internal ear. He did not have spontaneous nystagmus. However, the accident occurred five days before this examination on July 15th, so if the nystagmus lasted only three days after the injury, it would not have been present at the time of the examination. With many fractures at the base of the skull we find a facial paralysis, because with a fracture of the petrous portion of the temporal bone we have involvement of the vestibule, cochlea and aqueductus fallopii. If only the tip of the petrous portion of the temporal is involved, the above three escape injury. He was able to hear a conversational voice at a distance of one and one-half meters with the left ear only. If he had only a slight fracture of the cochlea, the endolymph

may have regenerated in five days with return of a moderate degree of hearing as a result.

Turning to the right ten times produced nystagmus to the left for thirty seconds, and turning to the left ten times produced nystagmus to the right for twenty-four seconds. This is a normal reaction of the labyrinth, so it is hardly necessary to test the internal ear with the tuning forks.

Concussion of the labyrinth cannot be demonstrated, so we should not make such a diagnosis; but fracture can be demonstrated both before and after death. (Please do not make a diagnosis of concussion of the ear or of the base of the skull. Ruttin.)

As this patient had a bandage under his chin and had lost several teeth as a result of the injury, we were forced to conclude that he fell on his chin and not on the top of his head. The force of the blow was transmitted from the inferior maxilla to the anterior wall of the external auditory meatus, resulting in a fracture of the latter. This accounts for the blood in the meatus. However, pus was present with the blood. He may have had either an acute or chronic purulent otitis media before the accident. (Ruttin thinks such cases should be operated upon, and he related a case to illustrate his point.) However, in the present case it is better to wait and pack the external meatus with gauze after dry cleansing, as the fracture, in this case, in all probability, did not involve the middle or internal ear.

Diagnosis.—Fracture of the anterior wall of the external auditory meatus. (The X-ray was to have been used, but the author did not learn what it revealed.)

Case 10.—A boy of six. He had acute purulent otitis media about eight months ago. Three weeks after the onset of the acute attack he developed sepsis, spontaneous nystagmus and vertigo with loss of vestibular reaction and hearing. His temperature at this time was 39° C., so that one was forced to think of either a purulent labyrinthitis or a sinus thrombosis. If he had a purulent labyrinthitis he would have a meningitis, and there were no meningeal signs or symptoms. On the other hand, we were not apt to find a purulent labyrinthitis in this case, as the otitis is acute.

Diagnosis.—Acute serous labyrinthitis.

Case 11.—Male, age thirty-four years. A history of chronic

purulent otitis media of the right ear for the past seven years, and of the left for the past year. Recently he has been having headache, tinnitus, nausea, vomiting, vertigo, etc.

Examination of the left ear revealed a dry (?) perforation which led into the antrum just posterior to the short process. He heard a conversational voice at a distance of one-fourth meter with the right ear and at two meters with the left. By Weber's test the sound was lateralized in the left ear. Rinné's test was negative for both ears, and bone conduction was lengthened for both. The hearing for fork C 1 was shortened, and for C 4 it was normal. Spontaneous nystagmus was absent.

In testing for the fistula symptom in the left ear, nystagmus was produced by compression, with the quick component to the left, and nystagmus to the right by aspiration, which is the typical reaction and is with the rule. The fistula test failed to produce a reaction on the right side. If we obtain only the slow component of the nystagmus by the test, it should be directed to the opposite side in the typical and to the same side in the atypical cases. In the absence of the central control (cerebral control), as under ether or chloroform anesthesia, we obtain only the slow or peripheral (vestibular) component. The fistula may be single or multiple. The horizontal canal is involved in ninety per cent of the cases in which suppuration is present in the antrum. In other cases the fistula may be present in the frontal canal, oval window, round window or cochlea.

Turning to the right ten times produced nystagmus to the left for thirty seconds, and turning to the left ten times produced nystagmus to the right for thirty-one seconds. This, of course, was a normal reaction.

As the vertigo manifested itself in this patient only fourteen days prior to this examination, and as his hearing was as good as it had been previous to this, he has only a fistula in the bony labyrinth, without a disease of the membranous labyrinth.

Diagnosis.—Circumscribed serous labyrinthitis. Therefore, in this particular patient only a radical mastoid operation is indicated, and not an operation on the labyrinth, too. However, as his hearing is poor in the other ear, we may treat him for awhile. Without treatment he is almost certain to develop a purulent labyrinthitis. We must attempt to close the fistula with either fibrous tissue or bone.

Case 12.—A boy of six years presented himself for examination. He gave a history of acute purulent otitis media in the left ear and of chronic purulent otitis media in the right ear. His head was inclined slightly to the right, and the right side of his neck was stiff. The hearing in the left ear was almost perfect, but was very poor in the right. The patient had been vomiting that day and was very dizzy. His temperature was 39° C. Further testing revealed destruction of the right labyrinth.

At this time we have to think of the following causes of torticollis: First, meningitis; second, cerebellar abscess; third, temporosphenoidal abscess, if far back in this lobe of the brain; fourth, sinus thrombosis; fifth, perisinus abscess; sixth, Bezold's type of mastoiditis; seventh, meningismus; eighth, endocarditis.

This patient's nausea, vomiting and vertigo may have been due to a cerebellar abscess. The temperature of 39° C. may mean a sinus thrombosis. With meningitis, the higher the fever the greater the torticollis. The destruction of the labyrinth may also explain the nausea, vomiting and vertigo. A sinus thrombosis would account for the fever and torticollis. Ruttin thought a sinus thrombosis combined with a serous labyrinthitis the most probable cause. Therefore, he performed a simple mastoid operation with sinus exposure. An abundance of granulations covered the sinus, but the sinus was not opened. Two days later the sinus was incised because the temperature continued high. At this time the internal jugular vein was also exposed, as it should be, and two infected lymph glands were found lying over the internal jugular between the thyroid and facial veins. This part of the vein had, in all probability, been infected by the suppurating glands. A resection of the internal jugular vein between these two contributing veins proved this to be the case, as a thrombus was found here and not in the lateral sinus.

Diagnosis.—Otitische lymphadenogene Leptico-Pyanne, or infection of the internal jugular vein from infected glands. The lymph glands around the internal jugular are in the chain from the middle ear and external auditory meatus. This patient made an uninterrupted recovery, but he is deaf in the right ear, because the right labyrinth was destroyed by a severe type of serous labyrinthitis.

Case 13.—For the past five weeks the patient has suffered with attacks of vertigo, nausea, vomiting, diarrhea, formication, tinnitus and deafness. The last symptom was present only on the left side. The membrana tympani was negative on each side and spontaneous nystagmus was absent. The functional tests showed the following: The conversational voice was heard at a distance of five meters with the right ear and at two meters with the left; the whispered voice at four meters with the right and not at all with the left. By Weber's test the sound was lateralized in the right ear, and Rinné's was positive for each ear. Bone and air conduction were shortened for the left ear.

Upon using the finger test the patient's finger deviated to the right of the observer's before the turning test had been made to the right and after turning to the left, but the deviation to the right was greater after turning to the right. Syringing the left ear with cold water produced a slight nystagmus to the right.

If a brain lesion were present in this case it must have been on the left side, as the left ear was the diseased one and the lesion was in the cerebellum.

Diagnosis.—Increased irritability of the labyrinth due to a disease of the left posterior fossa or left cerebellar lobe, but we do not know the exact nature of the lesion from the data which the case affords.

Case 14.—A woman of twenty-two years was admitted with the following history: Two weeks before her admission to the hospital she developed a full feeling in the right ear, followed in three days by severe, deep, boring, constant pain. A free incision was made in the right membrana tympani the day the pain developed and in three days the pain had ceased. The day before her admission, or thirteen days after the onset of the disease, she developed severe pain in the right middle ear and mastoid process, immediately followed by nausea, vomiting and vertigo. Her temperature on the day of admission, at 10 a. m., was 37.6° C.

Examination of the right ear revealed a red membrana tympani with pulsating pus filling a perforation in the anterior inferior quadrant. The mastoid was tender, upon deep pressure, over the antrum and tip. The left ear was negative while the right was quite deaf. Rinné's test was negative for the

right ear, but fork C 1 was not heard. The hearing for fork C 4 was shortened. The right labyrinth gave a normal reaction to the caloric test. In addition, the patient had diffuse bronchitis and was in the sixth month of pregnancy. There was also a periarticular abscess above the left hip joint.

During the subsequent twenty-four to forty-eight hours the mastoiditis underwent resolution, due to the application of cold, etc. However, the fever, bronchitis and periarticular abscess continued, so that it was necessary to think of a lateral sinus thrombosis with metastases in the lungs and hip joint, even though the mastoiditis had undergone resolution.

On the third day an exploratory operation was performed and the lateral sinus was exposed. Although the right ear was quite deaf, there was an absence of spontaneous nystagmus and the labyrinth responded normally to the caloric test. The deafness was, in all probability, toxic, so there was not any indication for a labyrinth operation. The lateral sinus was found far forward and was exposed above, but was perfectly healthy. Ruttin did not feel justified, under the existing circumstances, in ligating the internal jugular and exposing more of the sinus. For the same reason he did not open the antrum, as he found both the mastoid process and lateral sinus normal. He prefers to leave the wall of hard protecting bone, in such cases, between the antrum and mastoid tip.

Diagnosis.—Osteopyogenea or pus following the venous blood into the circulation.

Case 15.—This patient was admitted to the hospital February 18, 1905, with a history of headache, coryza, bronchitis and acute purulent otitis media of the right ear during the two weeks previous to admission. The mastoid was tender upon deep pressure and the right membrana tympani red and perforated. The functional tests showed only a middle ear disease, as the labyrinth was normal. The temperature was elevated. As the fever and other symptoms of mastoiditis continued for thirty-six hours, in spite of the abortive treatment, a simple mastoid operation was performed. The patient made a good recovery following the operation.

In February, 1911, the patient discovered pus discharging through a retroauricular opening. The discharge continued with more or less irregularity until he came back to the hospital in July, 1911. Two days before admission (July, 1911)

he suffered from vertigo, nausea, vomiting and chills. The functional tests and the physical examination showed only an acute exacerbation of a chronic purulent otitis media.

In this case we have to think of an old circumscribed serous labyrinthitis since the operation in 1905, because upon closer questioning we find that he had had vertigo since two months after the operation. The granulations over the horizontal or external semicircular canal may have destroyed the outer wall of the bony labyrinth.

In July, 1911, a radical mastoid operation was performed by one of the youngest assistants in the ear clinic, but unfortunately the operation was followed by facial paralysis. A great many large cells were found in the mastoid, filled with pus, but the dura and lateral sinus were not exposed. For four days after this second operation the patient ran a septic temperature, so that on the fourth day a third operation was performed for exploration of the dura and lateral sinus. The dura was negative upon pressure, but the jugular bulb was covered with granulations. These were easily wiped away with a piece of sterile gauze, and the blood flowed freely from an accidental tear in the wall of the bulb inflicted by the gauze.

After the third operation the septic temperature continued, but was higher than before, without even returning to normal at any time during the twenty-four hours. This meant, of course, a complicated case of sepsis. For instance, erysipelas complicating sepsis of aural origin will produce such a temperature. A septic temperature due to a lateral sinus thrombosis only always drops below normal before each rise. We must think, too, in this connection of meningitis as a complication. However, further examination and investigation revealed an abscess in the region of the left tibia.

At the time of the third operation the right internal jugular should have been ligated, as after this the shock was too great to permit a fourth operation even under a local anesthetic.

Diagnosis.—Mural thrombosis in the lateral sinus and possibly a brain abscess.

Case 16.—This patient, a man of fifty years, complained of inability to hear well for the past twenty-five years, but he has always considered the left the better ear. For many years he has had tinnitus of both ears. Three or four months before his admission he developed vertigo, but this symptom ceased

after taking potassium iodid. Fourteen days before this examination the left ear suddenly became worse. He admitted having had gonorrhea, but denied syphilitic disease.

The functional tests showed absolute deafness in the left ear and almost total deafness in the right. Spontaneous nystagmus was absent. Turning to the right ten times produced nystagmus to the left for thirty-two seconds, and turning to the left ten times produced nystagmus to the right for twenty-seven seconds. The finger test was negative. The right ear failed to react to the caloric test, and the left reacted a very little, if any.

Upon further inquiry the patient stated that he was in the habit of drinking five liters of beer a day and of smoking three cigars. Before he became totally deaf he was immediately made worse if he drank and smoked.

Diagnosis.—Toxic neuritis of the cochlear division of the auditory or eighth nerve. (Alcoholic neuritis.)

Case 17.—A boy of seventeen years complained of loss of hearing for the past two years with vertigo, but without tinnitus. His deafness was accompanied by epileptic seizures from the first, and his eyes showed a slight indefinite ocular nystagmus.

He heard only a loud conversational voice with each ear, but was unable to state with certainty in which ear he heard the fork better by Weber's test. Rinné's test was negative for each ear, and the bone conduction was shortened. It was doubtful whether he heard fork C 1 with either ear, and he heard fork C 4 only a very short time. He heard a conversational voice through the two and one-half meter speaking tube with each ear. Each membrana tympani was normal.

Since he had an indefinite ocular nystagmus, it was necessary to observe this carefully under smoked or colored glasses before making the turning test, so as to recognize any change in the nystgmus after the turning. Turning to the right ten times produced nystagmus to the left for thirty-nine seconds, and turning to the left ten times produced nystagmus to the right for forty seconds. The finger test was important, too, in this case, because he had a disease of the brain (epilepsy). The test was made, but was normal.

Diagnosis.—Otosclerosis of each ear. The increased irritability of the labyrinth was due to epilepsy, and the disease of the cochlea to the otosclerosis. The boy had congenital am-

blyopia (reported from the eye clinic), ocular nystagmus and epilepsy, so he is a degenerate.

However, the family history, so far as the ears are concerned, was negative.

Case 18.—A young lady of twenty-three years came to the clinic, at the request of her family physician, for an examination of her ears. She insisted that her hearing was perfect, but her physician felt an examination was necessary. She gave the following history: Two years ago she had an attack of optic neuritis in the left eye, which yielded to treatment with potassium iodid. Three weeks ago she had a recurrence of the inflammation in the same eye. She complained of a severe headache throughout the entire head, which she could not localize at any point. In addition, she had intestinal catarrh.

The physical examination of her ears revealed a normal ear drum on each side. The functional tests showed the following: The conversational voice was heard at a distance of ten meters and the whispered voice at five meters with each ear. Rinné's test was positive for each ear, but Weber's was indefinite. Bone conduction was normal, and she heard forks C 1 and C 4 perfectly. Spontaneous nystagmus was present and was most pronounced when she looked to the left. Her nystagmus was purely horizontal and was central, but a combined horizontal and rotary may be either central or vestibular or both. As it would be very hard to tell about a horizontal nystagmus produced by turning with the head erect, in this case the caloric test was used, as this test produces rotatory nystagmus. After syringing the right ear with cold water she had a rotatory nystagmus while looking straight ahead. However, the horizontal nystagmus did not disappear. With central nystagmus we cannot tell whether it is first, second or third degree. When central nystagmus is present the movement is quite pronounced upon looking either to the right or to the left, but it is absent while looking straight ahead. With central nystagmus we may have the third degree without the first or second, which is impossible with a vestibular nystagmus. In other words, with a third degree vestibular nystagmus we always have the first and second degrees. This patient vomited spontaneously and without nausea just before the syringing, but not during or after the syringing, so we are dealing with a central lesion. Cases of vestibular nystagmus are more irritable and more liable to

vomit during or after the syringing. The central cases are irregular and the vestibular ones are regular. This is true because brain tumors develop slowly, and nature has an opportunity to adapt herself to the slowly increasing intracranial pressure up to a certain point. The left ear failed to react to cold syringing.

If she stood erect, with her feet together and her eyes closed, she fell to the right; if she tried to walk a straight line, she deviated to the right. While standing erect with her head turned to the left, she fell forward. In all positions except one she fell to the right of her head. Changing the direction of the quick component from one side to the other is indicative of a brain lesion. Nystagmus due to a brain tumor (central) is apt to increase in severity, while that due to a vestibular disease diminishes. For instance, a cerebellar abscess will suddenly increase the nystagmus. The changeable character of the nystagmus is indicative of a brain lesion. In this patient, at one time the nystagmus was rotatory, at another horizontal, and later vertical. Turning to the left produced nystagmus to the right for seventeen seconds, while during the turning she had strabismus.

When one labyrinth has been entirely destroyed the other becomes compensatory. Therefore, the caloric reaction is lost on one side, but it is present on the other, so that the turning test is normal. The finger test was negative. It may, however, change at any time to the abnormal and then back to normal again. At one time she falls to the right, but at another she may fall to the left. The galvanic test was negative. The corneæ were sensitive and the facial nerves normal.

Diagnosis.—Cerebellar tumor. It is possible that the tumor was farther forward, but it exerted pressure upon the cerebellum.

Case 19.—A man, age forty-five years, presented himself, July 26, 1911, for examination, with the following history: In 1898 his head was run over by a carriage, and the next day he had nausea, vomiting, vertigo, tinnitus and deafness. His hearing grew steadily worse, so that in two months he was entirely deaf in the right ear. In 1909 he thinks he had middle ear catarrh. Ever since the accident he has had, from time to time, a swelling, with pain, behind the right ear.

The examination of the hearing revealed the following: The right ear was totally deaf. With the left he heard a conversational voice at seven meters and the whispered voice at two to three meters. By Weber's test the sound was lateralized in the left ear. Rinné's test was positive for the left and infinitely minus for the right, as he heard the fork when held over the right mastoid only in the left ear. The bone and air conduction were both shortened for the left ear, although he heard forks C 1 to C 4.

Examination of the eyes showed an absence of spontaneous nystagmus. The right ear was then syringed and produced nystagmus to the left, which was normal. Turning to the left ten times produced nystagmus to the right for twenty seconds, and turning to the right ten times produced nystagmus to the left for fifteen seconds, which is normal. The finger test was normal after turning to the right, but after turning to the left the patient either struck the observer's finger part of the time or passed to the right of the observer's finger. (Bárány has seen a few cases which are atypical, and this case is atypical.)

This patient's deafness was due either directly or indirectly to the traumatism. If directly, it must have been due to a fracture (basal) or a hemorrhage or both. If indirectly, to atrophy or degeneration of the eighth nerve. The cochlear division of the eighth is more susceptible to toxins and trauma than the vestibular. This patient had the symptoms of a basal fracture for three weeks after the injury, so the cochlea or cochlear nerve must have been involved. Possibly there was also some degeneration of the vestibular nerve. This can be determined by the galvanic test of both ears simultaneously or by syringing both ears simultaneously. Syringing both ears simultaneously with cold water produced in one-half minute nystagmus to the right, showing that the right vestibular apparatus was less irritable than the left.

Diagnosis.—Basal fracture involving the right side in 1898, with subsequent atrophy of the eighth nerve on the right (cochlear division entirely and vestibular partly).

BIBLIOGRAPHY.

1. **"Researches on the Rhythmic Nystagmus Reflected from the Vestibular Apparatus of the Ear and Its Accompanying Symptoms." Monatschrift für Ohrenheilkunde, 1906.**

XXXVI.

SOME CONDITIONS ASSOCIATED WITH THE LOSS OF CEREBROSPINAL FLUID.*

By R. Bishop Canfield, M. D.,

Ann Arbor.

I wish to relate to this society the following cases, during observation upon which, either before or after operation, there appeared an escape of cerebrospinal fluid, and to draw some conclusions from them.

Case 1.—Mrs. Lizzie A., aged fifty-two years, Jackson, Michigan. Patient presented herself on account of a suppurative otitis media on the left side, with dizziness. During childhood she had had scarlet fever, since which time the ear has discharged at intervals. Four years ago she had a mastoid operation in San Francisco. The ear and mastoid healed after about one year's postoperative treatment. Patient remained well for some months, following which occasional suppuration has been noted. About three weeks ago swelling appeared over mastoid. Since this time she has been dizzy, staggers to the right, and has been frequently nauseated.

Examination of the left ear showed great destruction of the tympanic membrane; tympanum contained small quantity of somewhat foul smelling, purulent discharge. Easily bleeding granulation tissue noted around the oval window. Mastoid scar infected, red and swollen.

Hearing test.—Conversational voice, not heard. Weber to the right. Rinné negative. Schwabach heard in the right ear. C-1 not heard. C-4 not heard. Nystagmus of one degree to both sides.

Turning test.—Turning to the right, after-nystagmus of fifteen seconds. Turning to the left, after-nystagmus of fifteen seconds. Caloric test, negative. Fistula test, negative.

---

*Read before the American Laryngological, Rhinological and Otolaryngological Society, Washington, D. C., May 10, 1913.

Radical mastoid operation. Mastoid showed the effect of previous operation, pus and granulation tissue in the antrum, middle ear and attic, easily bleeding granulation tissue about the oval window. Footplate of the stapes in position, although very loose. Labyrinth not opened. Good recovery from the anesthetic.

For eight days after the operation patient showed a spontaneous rotatory nystagmus to the right, which gradually disappeared. During the after-dressings the stapes could not be seen. During this time there was an oozing of clear fluid from the oval window. This reappeared as fast as it could be mopped away. At several dressings it was observed long enough to make it certain that more fluid appeared than could have been contained in the labyrinth, consequently it was concluded that this must be cerebrospinal fluid. This escape of fluid persisted for about ten days, after which it ceased. Recovery uneventful.

Case 2.—Mrs. Polly R., age forty-six years. Patient was referred to the clinic of otolaryngology on account of a right suppurative middle ear disease. She had had measles forty-two years ago, since which time her ear had continued to discharge. About seventeen years ago had periodic attacks of dizziness. At this time several polyps were removed, which operation relieved her of her vertigo. For many years she has had pain on the right side of the head in the parietal and mastoid region. She suffers from occasional attacks of vertigo and feels like falling to the right. Two weeks ago had a severe chill.

Examination.—Conjunctivæ both sides anesthetic. Pharynx slightly anesthetic. Right ear, marked destruction of the tympanic membrane, tympanum contains foul smelling pus, mastoid slightly tender to pressure.

Hearing test.—Conversational voice, not heard. Weber to the left. Rinné negative. Schwabach heard in the opposite ear. C-1 not heard. C-4 not heard. Spontaneous nystagmus, none.

Turning test.—Turning to the left, after-nystagmus of twelve seconds. Turning to the right, after-nystagmus of twelve seconds. Caloric test negative. Fistula symptoms positive.

July 5, 1912. Radical mastoid and Hinsberg labyrinth oper-

ation. Superficial sclerosis. Deeper parts of the mastoid, antrum and middle ear filled with pus and cholesteatoma. No fistula through canal system noted. Stapes absent. Oval window covered by elastic membrane resembling scar. Upon opening the vestibule there was free hemorrhage; granulation tissue not seen.

July 17, 1912. Patient complains somewhat of dizziness. Horizontal and rotatory nystagmus to the left of one degree. Two days later nystagmus had disappeared.

July 28, 1912. Clear fluid escaping from the labyrinth. This fluid of sufficient quantity to preclude its formation in the labyrinth. Discharge persisted for about ten days.

August 24, 1912. Patient well.

Case 3.—Mrs. Sarah V., age forty-three years, Ann Arbor, Michigan. Patient presented herself in the clinic of otolaryngology with a history of having had a discharging ear since childhood. She has occasionally had severe pain over the left mastoid and left side of head, and has had occasional attacks of dizziness, during which she has not fallen, but has felt great discomfort. Examination of the left ear showed the deeper parts of the canal filled with foul-smelling crusts and pus, with some thickening of the anterior inferior canal wall and great destruction of the tympanic membrane. Shrapnell's membrane, posterior to the short process, was lacking; stapes and incus plainly seen through perforation. After considerable nonoperative treatment without improvement in her condition, a radical mastoid operation was advised. This was performed April 27, 1909. A tedious but satisfactory convalescence was established and the patient made a satisfactory recovery.

October 28, 1909. Since the radical mastoid operation there have been times when the ear would discharge somewhat. During the periods of discharge she felt perfectly well, but between attacks complained of some dizziness and pressure in the head. Examination of the ear at this time showed it to be completely epidermatized.

January 31, 1910. For several weeks the patient felt perfectly well and gained in weight; ear has remained dry. Recently she has had some dizziness and pain in the head and sense of discomfort in the left eye. There is a slight horizontal nystagmus of one degree to both right and left. From this

time on the patient was treated occasionally in this and the neural clinic.

October 14, 1911. Nystagmus of one degree to both right and left. Turning to the right showed after-nystagmus of twenty-two seconds; turning to the left showed after-nystagmus of forty-five seconds, with marked dizziness. Irrigation of the left ear with cold water showed slight horizontal and rotatory nystagmus when eyes were turned to the opposite side, lasting a very few seconds, without dizziness. Irrigation of the right ear with cold water showed horizontal and rotatory nystagmus to the left, lasting one minute and fifty seconds, with marked dizziness.

October 17, 1911. Turning to the right gives an after-nystagmus of twenty seconds with marked vertigo. Turning to the left, no after-nystagmus and very slight vertigo. Irrigation of the left ear with cold water gives a slight horizontal and rotatory nystagmus to the opposite side. Patient stone deaf. Leucocytosis, 10,500.

November 1, 1911. The following operation was performed under ether: Old mastoid wound opened up, found perfectly epidermatized. Wound cavity discovered as at previous operation. As the operation progressed there was discovered a sac capable of holding probably one-half dram of material, with an opening into the tympanic end of the eustachian tube. This sac was curetted away. The cerebellum was uncovered and an attempt made to open the labyrinth from behind. On account of the escape of cerebrospinal fluid from a small circular opening in the dura, which was probably a large perilymphatic duct opening atypically into the subdural space, this stage of the operation was not permitted. The labyrinth, however, was opened widely from in front. As a result of the packing necessary to control the flow of the cerebrospinal fluid, the opening in the dura was much enlarged. One stitch was then taken in the dura. The patient made a good recovery from the anesthetic.

November 3, 1911. Following the operation the patient's subjective symptoms improved remarkably, and she said that she felt better than for a long time. She developed a slight nystagmus of one degree to the opposite side, which persisted for three or four days. On the second day following the operation the amount of cerebrospinal fluid lost increased so

markedly that the bandages, pillow and upper part of the bed were soaked for three days. The patient developed a temperature, stiff neck, Kernig's, Babinski, edema of the retinæ and some opisthotonos. She became somewhat stuporous. However, a leucocytosis did not develop. It was apparent that the patient was rapidly growing worse and without some further operative interference would probably die shortly. On this account the following operation was done at midnight: The dura of a dog was secured and tamponed firmly against the cerebellum in such a way as to cover the exposed cerebellar tissue and compleetly close the opening, which at this time was about the size of the middle finger. Following this procedure the patient was put in bed with the head of the bed elevated. Following this operation the cerebrospinal fluid ceased to escape, and there was a marked improvement in the patient's subjective condition with decrease in pain and decrease in temperature.

November 7, 1911. Despite occasional attacks of severe occipital and right-sided headache, and the fact that her temperature ranges from 101 degrees to 104.6 degrees, the patient's subjective condition continues to improve. Today patient had a slight nose bleed. The wound was dressed and found perfectly clean, the dural graft was in position and there was no escape of cerebrospinal fluid. A Widal test was ordered and was negative.

November 9, 1911. For each succeeding day the highest temperature is lower than the day before, the highest for today being 102.6 degrees. Last night patient complained of pain in the epigastrium and fainted. An ice cap was placed over the heart and a turpentine stupe over the abdomen. The patient slept very little. She feels fairly well today. The wound was dressed and found perfectly clean. Although there was some discharge on the dressings, there was no evidence of the escape of cerebrospinal fluid, and the dural graft was in position. The white blood count was 6,500.

Then followed an uneventful convalescence.

Case 4.—Karl S., age twenty-three years, Kalamazoo, Michigan. Patient was referred to the clinic of otolaryngology on account of a discharging ear. He was well until six months ago, when he became rather suddenly dazed and stupid. He continued in this condition for about three weeks, without

much fever or other symptoms than this mental terror. After this he made a gradual improvement and gained considerably in weight. After about two months he was able to look for work, although he did not take a position. About six weeks ago he took cold, at which time the ear began to pain. Pain in the ear and discomfort in the right side and back of head continued until this time. During this time has also complained of occasional severe frontal headache. A week ago the ear began to discharge a clear fluid. Five days ago complained of severe frontal headache in the evening, and on the following morning was found unconscious. He remained unconscious for twenty-four hours, and since then has been semiconscious and has complained of great pain in head and neck.

Examination.—He appears stuporous. Answers questions indifferently well and slowly. He knows his name, but not where he is. His neck is stiff. Double Kernig's and Babinsky. Patellar reflex absent. Pupils dilated. Nerve head and fundi slightly fluffy. Double facial paralysis, more marked on the left side. Leucocytosis, 28,000. Urine shows large quantity of albumin and many casts. Cerebrospinal fluid is purulent and contains many streptococci. Does not reduce Fehling's solution. Right ear concha is filled with perfectly clear fluid, deeper parts of canal contain pus. A clear fluid is seen escaping from the Rivinian segment. It pulsates rapidly and is apparently cerebrospinal fluid. Tympanic membrane is dark and retracted in the anterior portion and bulging in the posterior half. Mastoid not tender. Tongue dry. Buccal cavity and tongue covered with crusts. Teeth covered with sordes. Drainage of the basal cistern was immediately made according to the method of Haynes. When the dura was opened, cerebrospinal fluid escaped freely. This fluid contained large floccules of pus. The base of the brain was irrigated with warm normal salt solution during the operation. The patient recovered from the anesthetic in good condition, although respiration was very slow and deep, six per minute. It was noted that immediately after the lumbar puncture, which was done before the operation, the ear ceased to discharge. Two days later the patient died.

Autopsy showed a plastic basal meningitis with extension along the fissure of Sylvius. The apex of the right temporal

bone was occupied by a very large pneumatic cell containing cerebrospinal fluid and pus. The perforation through the wall of this cell had permitted the entrance of infection to the subdural space and the escape of the cerebrospinal fluid externally into the pneumatic structure of the temporal bone, thence to the middle ear and external canal.

Case 5.—William S., aged forty-two years, Kalamazoo, Michigan. Patient was referred to this clinic on account of a carcinoma of the temporal bone which had been present for about a year. Examination showed an extensive squamous cell carcinoma, involving the external canal, middle ear and mastoid, and which had perforated through the mastoid cortex to involve the soft parts. The labyrinth did not react to the physiologic tests.

December 27, 1912. Radical mastoid operation was performed and a large mass of carcinoma removed, together with the auricle. Patient made a good recovery and the wound became clean.

February 5, 1913. On account of the appearance of a spontaneous nystagmus of the third degree to the diseased side and marked disturbance of the equilibrium, it was decided to resect the temporal bone. This was accordingly done. It had been the intention to remove the affected meninges and to do a dural transplantation, but inasmuch as it was discovered during the operation that the disease was too extensive, this transplantation was not made. During the operation and for some days afterwards there was a free discharge of cerebrospinal fluid. This, however, ceased and the patient made a good recovery. He was discharged to the care of his family physician to die a month or two later as a result of the recurrence of the growth.

Case 6.—Elizabeth M., age four years, Frankenmuth, Michigan. Patient was referred to the clinic of otolaryngology from the neural clinic with the following history: Nine months ago she fell on a toy boat, the mast of which was made of a knitting needle. The needle entered the orbit just below the eye. A piece of the needle two inches long was removed immediately and the wound healed. It did not affect the eyeball or vision apparently. The wound healed within a few days. After that she complained of headache, vomiting, more or less constant sleeplessness, and about four weeks after, an

incision was made at the inner side of the eyeball, from which pus escaped. Six weeks after that Dr. Slack of Saginaw operated upon the orbit and removed another piece of steel about two inches long, which showed blunt and broken ends. The parents believe that the needle was broken off before the accident happened and that this part is the end of the needle, but this is not absolutely certain. The child remained well after this part was removed until about seventeen days ago, when she began to complain of headache, sharp and piercing in character. She vomited and was sleepless. No dizziness nor staggering was noted. When the trouble began seventeen days ago she complained of pain only in the left frontal region, but now the pain is on both sides. She vomits less frequently now than formerly, and has slight fever. Temperature has been moderate, from 99° to 100° and 101° or more in the evening. She has not had any cross eyes in the last seventeen days. There is no rigidity in the back of the neck. She is irritable to noises and light, but apparently not excessively so.

Neural examination: Patient is able to sit up, whines constantly, has an expression as if in pain. Says that she is in pain, but does not know where it is. She responds to questions, but evidently does not understand fully. Head: The pupils are equal and react to light and accommodation. There is no strabismus nor extraocular palsy. No tremor or atrophy of the tongue, no facial paralysis, no fever, no rigidity or retraction of the back of the neck. There is a small scar just outside the internal canthus of the left eye in the lower eyelid, slight ptosis. No Kernig or Babinsky. Chest and abdomen negative. Urine negative. Blood: Red, 5,125,000; white, 16,000; hemoglobin, 70 per cent.

December 10, 1910. Transferred to this clinic for examination and operation. Ear, nose and throat examination negative.

Operation.—Usual frontal sinus incision through the left eyebrow to the median line. Vertical incision upward two inches long in the median line, periosteum retracted, cortex negative. Frontal sinus opened and found negative. Entire frontal wall of frontal sinus removed. Dura uncovered one inch in diameter. Dura bulged strongly forward, marked pulsation. U-shaped flap, base upwards, one-half inch in diam-

eter, followed promptly by small brain hernia. Brain deeply incised in all directions with negative result. Dural flap sutured with catgut. Skin incision sutured for primary union. Good recovery from ether. For a week following the operation the patient was in good condition, pain on the left side of the head slightly greater than before operation. Temperature normal.

December 23, 1910. Having been restless for the past two or three days, on this morning she suddenly lasped into semi-consciousness, from which she could be aroused with difficulty. Both optic discs were swollen.

Operation.—Original incision opened, dura found bulging strongly; area of bulging was incised and a large amount of creamy pus escaped. Following the escape of pus clear fluid escaped from the depths; no doubt cerebrospinal fluid. For ten days following the operation both pus and pulsating spinal fluid escaped at the time of dressing. The wound was dressed daily with a Whiting brain searcher.

January 16, 1911. For sixteen days the wound has been clean, and no cerebrospinal fluid has escaped; that is, the ventricular fistula has closed.

Operation.—Wound edges freshened and sutured. During this operation it was noted that the dura bulged forward through the opening in the skull the size of a small hazel nut. It was very elastic and thin, and there seemed to be some fluid behind it. On account of the absence of symptoms and the fact that the brain had been searched carefully on several occasions since bulging had taken place, the brain was not opened.

January 19, 1911. For two days following the operation the patient remained in good condition, but on the following day had a rise of temperature, became very irritable and showed a leucocytosis of 21,000. Her disposition changed suddenly and completely; she became irritable and showed violent fits of temper. She complained of severe headache on the left side of her head. On account of these symptoms the skin incision was reopened and the brain incised, brain searcher introduced and a second abscess discovered at a somewhat deeper level than that occupied by the first one. Immediately following the operation the patient's condition changed entirely and she became happy and contented. Her convalescence was uneventful, except for an attack of nasal coryza.

This case is of considerable interest because of the fact that the abscess was a double one and could be clearly demonstrated as such. Again, it is a very rare thing to have a deep brain abscess recover. It is also interesting to note the effect of the abscess on the child's disposition. When she was brought in for the second operation she was the picture of a very naughty child with a violent temper; she cried and struck at her nurse; but after the abscess was drained and immediately upon coming out from her anesthetic, she showed a very sweet and lovable disposition. Since this operation she has been perfectly content, happy and obedient. At the present time she has a slight divergent strabismus and a slight regularity in her gait.

The method of conducting the after-treatment of this brain abscess may be worthy of mention. The abscess was neither packed nor irrigated, but was investigated daily by means of the Whiting brain searcher, an instrument which permits one to examine thoroughly every part of the abscess cavity, wipe it out gently and in this way by means of reflected light to control satisfactorily the healing process.

Case 7.—Glenn S., age eighteen years, Paw Paw, Michigan. Patient was referred to the clinic of otolaryngology on account of discharging wound in forehead and his mental condition. Patient well and strong until the present illness. Two months ago he had a head cold with free discharge of matter from the nose. During this time complained of great headache and sleepiness. Patient would answer questions, but would drop off to sleep during conversation. Slept most of the time. This condition of drowsiness lasted for two or three days. Patient not in bed, but apparently weak. Felt better and walked four miles. Then took an eighteen mile drive to visit his brother. During his visit complained of headache, chiefly in the left frontal region. At times would appear normal and then again stupid. Nose ceased to discharge. Remained home for about three weeks, gradually getting weaker and complaining continually about the pain in his head. His father noticed that he was somewhat confused in his speech. He frequently said that he was feeling "bigger," no doubt meaning that he felt better. He began to complain that he was seeing double, and the father noted that the left eye turned in toward the nose. He was taken to a hospital,

where an operation was performed. After the operation he made some improvement, his eyes became straight and his mental condition improved. Three weeks before his admission into this hospital he was again anesthetized, and an intranasal operation was performed for the purpose of securing drainage from the left frontal sinus. He continued to improve and was finally sent to his home physician for observation. During his residence in his home hospital he was not dressed by his physician, but was cared for by the nurse, from which it may be thought that his condition was not considered serious. After returning to his home the wound in his forehead did not heal, but continued to discharge foul pus. His mental condition did not arouse comment. On the night previous to his coming under my observation he began to show mental derangement; for example, when his father attempted to give him an enema, the patient placed the tube in his own ear. Later in the evening he becam stupid, his answers became delayed, and he again spoke of feeling "bigger" instead of better.

When he was brought to the University Hospital he was somewhat emaciated, but was strong enough to assume the active position in bed and to turn towards the examiner without assistance. He showed no paralysis. The knee jerk was slightly increased on the right side, and there was slight evidence of a Babinski's sign on that side. He also showed a double optic papillitis, more marked on the left side. His breath was foul, and the same foul odor was noted in the perspiration. He knew his name, his home and where he was, but in other matters his memory was defective. He answered questions intelligently but slowly, and always in complete sentences. If asked if he had pain, he replied, "I have no pain," etc. As soon as he had answered he would close his eyes and sink into a sort of stupor.

A sinus was present in the center of the left eyebrow, through which foul pus escaped. A probe passed gently into the sinus discovered no bone. On account of the location of the suppurative process and the patient's mental condition, a diagnosis of probable left frontal lobe abscess was made and an immediate operation advised.

May 25, 1910. Operation.—A U-shaped flap was turned up from the left forehead. The anterior wall of the left frontal

sinus was absent, and the frontal sinus was found filled with foul pus and prolapsed brain tissue. An opening through the posterior wall of the frontal sinus, about one-half inch in diameter, was discovered. Over this area the dura was gangrenous, while the bone in the neighborhood was carious. This condition rendered the formation of an osteoplastic flap impossible. The inner table was then removed until healthy dura was uncovered in all directions. To do this it was necessary to uncover an area about one and one-half inches in diameter. A sinus was discovered through the dura at a point about one-half inch above the center of the eyebrow. Through this sinus pus escaped. On account of the condition of the dura, a dural flap was impossible; therefore the brain was freely incised to permit the escape of indescribably foul pus, the odor of which must be experienced in order to be appreciated. This pus continued to escape for about fifteen minutes. During this time a very small amount of anesthetic was needed, and the pulse increased about twenty beats per minute. The right frontal sinus was opened and found to be filled with foul pus and necrotic tissue. The frontal sinuses were packed with iodoform gauze, the brain cavity not being packed. No sutures were taken. The patient regained consciousness on the table and was returned to bed feeling better than before the operation.

At the second dressing about one and one-half ounces of cerebrospinal fluid escaped from the depths of the wound, and from them on daily for a week cerebrospinal fluid was seen to escape, at the end of which time the ventricular fistula healed. He died three weeks after the operation without symptoms of meningitis.

Case 8.—Mr. B., age thirty-five years, Detroit, Michigan. Patient was referred to the clinic of otolaryngology from the surgical clinic, where he was being treated for discharging sinus near the median line in the left frontal region. Present trouble began nine months ago, at which time he struck his head on the fitting of a steam pipe. Later had pain and spontaneous rupture of the skin, with free discharge of pus. At this time the skin was freely incised at about the midpoint of the left frontal sinus, and since that time he has had more or less persistent purulent discharge. Patient states that nine years previous to this he had an accident while in a lumber

camp, which resulted in unconsciousness for forty-seven days and necessitated trephining. Present examination showed the left eye missing (due to the accident nine years ago above mentioned). Deep crater-like scar over the midpoint of the left frontal sinus, at which point patient had been trephined, and median to this is a small granulating sinus from which escapes thick pus. The throat is negative except for chronic pharyngitis. Nose shows small sharp spur low down on the right side, high deviation and small dislocation of the cartilage on the left side. No pus noted in nose. Transillumination showed right frontal sinus to transmit light freely; less light is transmitted on the left side. Ethmoid regions on both sides transmit light freely. Transillumination of the antra normal, light reflexes and sclera being obtained on both sides. Radiograph shows shadow inside the cranium at the level of the frontal sinus. Physical examination was negative.

October 10, 1912. Operation.—Incision through the left eyebrow with vertical incision upward to neighborhood of hair line. Removal of the anterior table showed frontal sinus filled with pus and granulation tissue. Brain uncovered for a considerable area corresponding to part of posterior wall of frontal sinus. Depressed fracture the size of thumb nail. Trephine opening over the frontal lobe lateral to and above frontal sinus, showing considerable new bone formation. Curettage of frontal sinus, removal of the entire anterior wall together with roof of orbit, discloses necrotic bone below area of uncovered dura, removal of which uncovered large area of the left frontal lobe and part of the right frontal lobe. Old rupture of dura noted over halfmoon-shaped area one-fourth inch in diameter, situated just below and in front of trephine opening. This rupture covered with very thin scar, which was torn during the operation, allowing the escape of a small quantity of cerebrospinal fluid. Wound irrigated and packed.

At the second dressing cerebrospinal fluid was seen oozing from the perforation in the dura near the outer angle of the wound. Wound otherwise clean and granulating. This escape of cerebrospinal fluid continued for four days, after which it ceased. Patient then passed through an uneventful recovery.

Case 9.—Mr. Van F., age fifty-two years. Patient was referred to the clinic of otolaryngology from the clinic of neurology, where he is being treated for right sided hemi-

plegia. Seven months ago patient fell fifteen feet, striking on his forehead. He did not lose consciousness, but walked to the house. Had a violent nose bleed, which lasted for two hours, at which time his doctor sewed up the wound in his forehead. About two weeks later he began to have difficulty in walking. This seemed to be due to weakness in his right leg. Later right arm became weaker, and he soon began to have difficulty in talking. Found that he could not remember the words he wished to use. Whenever he sits up, clear fluid to the extent of two ounces escapes from the left nostril, at which time he has left-sided headache. On several occasions, during which cerebrospinal fluid has been lost, he has become unconscious.

Neural examination (Dr. Camp): Patient's station with eyes open is fairly good, swaying slightly; but with eyes closed the swaying is very marked. The gait shows ataxia in the right leg, so great that he can only walk three or four steps without support. There is a slight right sided facial palsy, shown by a weakness in wrinkling the forehead on the right side, a slight aperture in the right eye when eyes are closed, and a lack of firmness in the right cheek when both are inflated. On protrusion the tongue goes slightly to the right. There is a very marked weakness in all movements of both right arm and leg. Dynamometer test with the right hand is 40, with the left 115. No ataxia is noted when the patient makes circles with the left leg, but with the right the movements are jerky and faulty. Some ataxia is shown in the right arm when the patient is asked to touch the end of the nose with forefinger. There is a subjective numbness throughout the whole right side of the body, but no anesthesia to touch can be demonstrated. Pain conduction is apparently delayed throughout the right side, as the patient says that he feels a pin point more keenly on the left side. There is no difference felt in the surface temperature between the two sides of the body. Reflexes: The pupils are equal and react to light and accommodation equally but rather sluggishly. The extraocular movements are normal. The biceps, triceps, kneejerks, and Achilles reflexes are all active on both sides, but more so on the right. The abdominal and cremasteric reflexes are present and about equal on the two sides. Well marked ankle clonus and a Babinski reflex are present on the right side, but not on the left.

Measurements: Upper arm at corresponding points, right, 24 centimeters; left, 27 centimeters; forearm, right, 22½ centimeters; left, 24 centimeters; thigh, right, 35 centimeters; left, 37 centimeters; calf, right, 27 centimeters; left, 29 centimeters.

Hearing test shows air conduction to be better than bone on both sides. The sense of smell is lost in the left nostril and diminished in the right, where he smells but cannot recognize accurately. He recognizes salt on both sides of the tongue, but does not recognize sweet or sour on either side.

The physical examination is negative, except for a marked depression, the size of a quarter, above the left eye, which is the site of the trephine done the night of the accident. There is some tenderness to pressure here, but no pulsation. The examination of the blood and urine is negative. Three days after his admission the eyes were examined in the ophthalmologic clinic and the following report received: Edematous retina. No hemorrhages nor papillitis. Convergence limited in the left eye. Upon sitting erect there was a loss of from one to one and one-half ounces of cerebrospinal fluid (Dr. Canfield). An exploratory operation was therefore decided upon.

July 31, 1909. Operation.—In the left frontal region U-shaped incision made and flap turned down, dissected away from adhesions about the old operative field and from the dura, which disclosed an opening in the bone one by one and one-fourth inches, from which perforation in dura was plainly visible, this perforation being three-eighths by one-half inch. Probe passed through this opening came in contact with brain; found brain collapsed so markedly that anterior surface of the frontal lobes lay one and one-half inches from dura. Dura incised and plastic done, closing the orifice by means of silkworm gut. Flap suture in place. The second day the stitches were removed and collodion dressing applied.

August 10, 1909. Patient in good condition, wound entirely healed, patient feels stronger, has strong grip in right hand, has had no more headaches and claims to be regaining control of right leg. About one month later patient resumed his duties as mail carrier, apparently perfectly recovered, although he was showing some signs of beginning general paresis (specific).

Case 10.—Mr. John F., age forty-nine years, Pontiac, Michigan. Patient was transferred to the clinic of otolaryngology

from the surgical clinic, where he had been operated upon twice for alveolar sarcoma of the neck.

Examination at this time showed tumor mass the size of a large fist, presenting in the right frontal region. This mass is fairly dense with soft areas. It involves the right upper eyelid, displaces the right eye markedly to the right and forward and downward, extends over the median line. Some edema of both left eyelids, also marked edema of the right lower eyelid. Shows a scar in the right upper eyelid, also a sinus in the right frontal region from which pus escapes. Mass extends upwards two-thirds of way to hair line, not attached to the skin, except at the two points mentioned. Nose shows the right nostril completely occluded by tumor mass, which is not attached either to the septum or the lateral wall of the nose anteriorly. Free purulent discharge from the nose. Neck shows several scars on the right side. Dense mass, the size of a large English walnut, not attached to the skin, lies under the anterior margin of the right sternomastoid. Left side of neck shows scars.

January 16, 1913. Operation.—Enucleation of a mass lying attached to the sternomastoid. Removal of one lobe of the submaxillary salivary gland which was lying in relationship with tumor.

January 18, 1913. Operation.—Under ether. Horizontal incision beginning at the outer angle of the left eyebrow and extending across to midpoint of the right eyebrow, and vertical incision in median line. Retraction of the soft tissues permitted the removal of a large mass of malignant growth. After this had been removed it was discovered that the outer table was lacking over the entire right half of the frontal bone and part of the left frontal bone. Tumor mass was scraped out of a large frontal sinus. Right frontal lobe occupied by the tumor to a considerable depth. Profuse hemorrhage, and operation was stopped at this point. Wound packed and incision sutured.

January 19, 1913. Wound dressed, escape of a considerable amount of clear cerebrospinal fluid from the deeper portion of the tumor; that is, from the lateral ventricle.

January 30, 1913. Daily escape of cerebrospinal fluid for four days, following which the escape ceased and the wound cleared up.

February 28, 1913. Patient discharged to the care of his home physician with evidence of tumor growth.

Case 11.—Patient, Alexander H., presented by courtesy of Dr. A. M. Barrett of the State Psycopathic Hospital, Ann Arbor, Michigan. Patient admitted August 20, 1906, suffering apparently from dementia. He appeared stupid and indolent, but responded accurately to questions. He is partially blind. Speech is thick, slurring, explosive and ataxic. During the past ten days patient has been noted holding his handkerchief over his nose, and when this was investigated it was noted that he had a serous discharge from the nose at the rate of about one drop per second. This fluid was found to contain a small percentage of albumin, fibrin and leucocytes. Cerebrospinal fluid was suspected. Death later from natural causes.

Autopsy.—Head Section: The surface vessels were enlarged. Upon removal of the brain the base of the skull showed marked signs of increased pressure, the cribriform plate being eroded and the brain substance protruding into the nasal cavity. The cella tursica is much enlarged, round and unusually deep. The middle cerebral fossæ are much deepened, and the floor in some places is eroded, due to pressure. The posterior cerebral fossa shows marked signs of pressure, being extremely irregular. On removing the cerebellum the inferior surface is seen to be unusually full, especially on the right side. The temporal and occipital regions of the left side of the brain are hollowed out as though from pressure. The aqueduct of Sylvius is dilated and small, dark granular mass protruding slightly from it. On the section through the upper part of the pons a small tumor mass 8 mm. by 9 mm. in diameter, is seen filling the right dilated aqueduct of Sylvius and greatly compressing the brachium of the same side. The lower portion of the sectioned pons shows the tumor mass completely filling the greatly dilated aqueduct. On the horizontal section through the vermiform process of the cerebellum, extending down to the fourth ventricle, the tumor mass, 2.5 cm. by 3.5 cm. in diameter, is seen filling the widely dilated ventricle, producing greater pressure upon the pons and medulla on one side and the cerebellar tissue on the other. The tumor is reddish gray in color, choroidal in type, in its arrangement looking like innumerable small vessels closely crowded together. Cranial nerves not well preserved, the infundibulum is greatly

dilated, being 12 mm. in diameter, and the transverse fibers of the chiasm are much less prominent than normal. Longitudinal section through the middle of the thalamus shows the lateral ventricles greatly dilated, especially on the right side, where the brain substance is seen bulging up in the posterior horn. The white and gray substance is greatly narrowed in all regions. Considerable injection of the fine vessels is seen. The ependyma of the lateral ventricles is coarse, granular and pinkish in color. The ependyma and brain tissue of the posterior horn on the right side seems to lie in folds. The pineal gland is cystic.

Sections from the tumor show it to be composed of massed loops and sections of tubes of cuboidal epithelial cells resembling proliferated choroid plexus tissue. In places there are cysts and masses of coagulated material. The tumor is evidently an epithelial tumor, springing from the choroid plexus of the fourth ventricle, and might be called an angioma or papillary epithelioma. The tumor by its location had caused an internal hydrocephalus which had produced a softening of the bones in the region of the base of the frontal lobes, and this explains the discharge of cerebrospinal fluid.

Cases Nos. 1, 2 and 3 are cases of labyrinth involvement occurring during a chronic suppurative otitis media. In case No. 1 the labyrinth was not opened at the time of operation, although failure to find the stapes after operation, together with the loss of cerebrospinal fluid daily for several days, are sufficient evidence of the fact that it had been opened.

In cases Nos. 2 and 3 the labyrinth was exenterated. Here are two cases in which the suppurative cavity was brought into communication with the subdural space without setting up a meningitis.

Case No. 3 is interesting in that it demonstrated the condition of meningismus into which a patient may lapse as a result of excessive loss of cerebrospinal fluid, and that a heteroplastic dural graft was successfully made for the relief of the condition.

Case No. 4 is interesting in that the patient showed evidence of meningeal involvement long before his attention was drawn to the ear condition, and also in that the avenue of infection of the meninges and the avenue of escape of the cerebrospinal fluid through the pneumatic structures of the apex of the temporal bone could be made out.

Case No. 5 is another case in which free communication between the subdural space and the suppurating cavity did not cause meningitis.

Cases Nos. 6 and 10, that is, the case of double frontal lobe abscesses, and the case of sarcoma of the brain, show how easily sudden decrease in cerebral pressure may cause rupture of the lateral ventricles into the abscess cavity without infecting the ventricle.

Cases Nos. 7 and 8 again are illustrative of the possibility of bringing the subdural space and an abscess cavity into communication freely without setting up a meningitis.

Case No. 9 is most interesting, as it demonstrates that hemiplegia may result from loss of cerebrospinal fluid, and that function may be restored by stopping this escape.

Case No. 11 (psyco.) demonstrates that the subdural space or base of the brain may remain in free communication for an indefinite period with the nasopharynx without establishing a meningitis.

XXXVII.

ODD CASES OF NASAL DEFLECTION WITH SUGGESTION AS TO TREATMENT OF NASAL ADHESIONS.

By Otis H. Maclay, B. S., M. D.,

Chicago.

The ordinary operation is always found to be interesting and instructive, but the slight or extensive departures from the original line of work found necessary by exceptional cases is, I believe, even more interesting, owing, first, to the original technic that must be developed, and, secondarily, to the peculiar formations present. It is with the hope of satisfactorily describing some of the oddities found in nasal deflections and also the technic employed in correcting the same that I have ventured this article. I believe in about all of the very decided and very irregular angular deflections that trauma in some form or other is the etiologic factor, differing in this respect from those that are of a more regular curve, although these may be deflections of extreme degrees. As an aid in describing these irregularities, I shall first briefly mention the anatomy of the nasal septum and the relation to it of the perichondrium and periosteum.

The bony septum is composed in the main of the perpendicular plate of the ethmoid above, the vomer posterior and below, resting upon the nasal crest of the superior maxilla, and the anterior portion of the superior maxillary crest, or the socalled incisor crest. This anterior extension of the vomer, the superior maxillary crest and incisor crest, by their blending, form the lower portion of the septum, and are commonly spoken of as the "ridge." Placed in the space left by the articulations of the bony septum we find the triangular cartilage. The coverings of the cartilage and bone form separate envelopes for each part. The perichondrium of the triangular cartilage, as it approaches the ridge, passes under

the cartilage to the opposite side of the septum, completely enclosing the cartilage. Similarly, the periosteum of the ridge passes passes over the bony crest to the other side of the septum, thus forming a complete envelope for the bone. These separate envelopes are closely united to each other at junction of cartilage and bone, and of course to the structures that they invest. This means with the ordinary deflection, which generally consists of cartilage and bone, that at least two separate envelopes must be opened before a complete removal of the deflection is possible. After having separated the necessary amount of perichondrium covering the triangular cartilage, and carrying the dissection to the covering of the bony ridge, we find we are unable to continue lower with blunt dissection without tearing the flap, since the area is reached where the perichondrium is turning under the cartilage and the periosteum over the ridge, going into the other side of the nose, and so at this point, the cartilage having been removed, it becomes necessary that the periosteum along the top of the ridge be incised and dissected from the bone down to the nasal floor. The union now of the periosteal layer below with the perichondrial layer above gives a large flap, extending from the desired line above to the floor, and when held by a retractor pulling laterally gives the appearance of a tent lying on its side, with the retractor acting as the crossbar at the top. Within these large flaps the bone can be readily seen and removed, either with chisel or bone biting forceps.

In order to demonstrate these points I have dissected a few septums. By examining them you will be able to see the relations of the various parts of the nasal septum to each other. These specimens, however, are used merely to aid the description, since they are not in most instances sufficiently irregular to warrant operative interference. With one exception the flaps have been dissected, not as done in the operation, but in order to demonstrate most clearly the various structures present. In specimen III an anterior view is given of the incision used and the freed flaps as seen while operating, with the exception of the right anterior inferior portion, where a fairly firm union is left as the cartilage rests upon the vomer. This shows how readily a tear could result if, without first freeing the flap at this point, the solid parts were removed. Specimen IV shows a portion of the perpendicular plate of the ethmoid

placed for forward, and extending downward to within one centimeter of the anterior extension of the vomer, completely overlapped, however, on the right side by the cartilage of the septum.

In some of the specimens that portion of the cartilage overlapping the vomer is shown. It is very essential, as you can see, that a piece of cartilage so located must be removed before the bony covering can be freed. After its removal the covered vomer comes into view, and an incision is made through the periosteum, formerly covered by the overlapping cartilage, to the bone, and the dissection carried downward as far as desired.

Among the most common irregularities met are those where the cartilage is split, but lying in its own plane. There may be several pieces of cartilage, each with its own envelope, and consequently each piece must be freed of its perichondrium before removal. Again, the cartilage may be so broken and the deflection so extreme that the septum presses against the outer wall of the nose, with the split some distance posteriorly. A case with a vertical and horizontal deflection illustrates this. The extreme of deflection was vertically placed, about one inch inside the nose, but the bend extended horizontally the length of the cartilage. At the greatest point of deflection (vertical) the anterior piece of cartilage was found completely separated from the posterior portion, the latter part having its own envelope, which consequently was opened and bluntly freed. In this case the anterior piece of cartilage was first removed and then, having more room and clearer view, the posterior dissection was completed.

An irregularity that has required considerable care in my hands is one where the cartilage, extremely deflected, bends forward well into the opening of the nose. Here we have blending of the skin and mucous membrane, with the mucous membrane very much thickened. We wish to keep our incision well inside the nose as an aid in healing, and also to prevent the pain felt by incising the skin. Consequently we place our incision as far inside as possible, and work the anterior covering of the deflection forward. This is sometimes quite difficult, owing to the thick fibrous tissue present. The mucoperichondrial covering of the concave side in these deflections is often extremely thin, owing to the constant removing of

crusts that form in the hollow of the cavity. Under these conditions, if the convex flap is torn in its freeing, great care must be exercised on the concave side, or a perforation might result.

Ordinarily the cartilage and bone occupy about the same relative space as the septum, but we find in exceptional cases a decided variation in the proportion of the two, and also in the thickness of the structures. In one case with a deflection extending posteriorly to a point opposite the middle of the middle turbinate, I was surprised to find the entire deviation consisting of the triangular cartilage, which was also very much thickened, measuring at the posterior portion at least one-fourth of an inch in width. Another case demonstrating the irregularity in amount of bone and cartilage showed the septum having a small anterior portion of cartilage, extending backwards for about one inch from the nasal opening, and the remaining portion of the septum being composed of perpendicular plates of ethmoid and vomer. This is the only one of my cases that has had the bone extending so far forward, although other cases have been reported.

In two cases, each having a moderate posterior deflection, but with a large, sharply pointed spur on the deflection, which extended well over to the outer wall of the nose, I removed both not only to give a better nasal condition, but because of the annoying tinnitus aurium present. The noise in the ears of both cases disappeared very shortly following the operation. Although appreciating the fact that there are many conditions producing such a disturbance, I mention this because of the result and not with the idea that tinnitus aurium is even likely to be benefited by such a simple remedy in all cases.

Among the various complications found in the septum work is a union between the outer and inner walls of the nose. This, when freed, results in two raw surfaces, with a tendency during the healing period of again uniting, since it is not uncommon to have considerable swelling for two or three days following the removal of the packing. In order to avoid such an unpleasant complication, I treat any denuded surfaces with thirty per cent silver nitrate immediately after removing the gauze. This is rubbed over the area; leaving the albuminate of silver for a covering eliminates the raw surface and consequently prevents later adhesions. I have employed this

solution for several years, not merely for the septal cases described, but have used it wherever I wished to prevent adhesions in the nose. The other methods, such as packing and the actual cautery, caused a decided reactionary swelling, and often the same condition recurs. With the actual cautery we have the reactionary swelling not only from the burning, but also from the congestion arising after the effect of the cocain has disappeared. I have been much pleased with the results from the strong nitrate solution, and take this opportunity of describing its application.

After cocainizing, the adhesion is broken up and then the denuded area rubbed over with thirty per cent silver nitrate, which is applied with cotton on the end of an applicator, care being taken not to have the cotton more than dampened with the solution, thus limiting its application to the region desired. This can be treated within two or three days again, but without cocainizing, simply applying a small amount of the silver nitrate. At this time considerable slough will be present, but this should not be disturbed until it comes away without bleeding, thereby insuring against a new raw surface. If bleeding occurs, however, the solution should be applied to the area involved.

Another condition of the septum that is out of the ordinary is where the anterior end of the cartilage is dislocated to one side, and almost protrudes from the nose, with the remaining portion of the deflection (the larger part) extending into the opposite nostril; that is, a horizontal "S." I believe in this class of cases, if the conditions are suitable, that the best results are obtained if two incisions are used. First the extreme anterior deflection is removed, and, the incision being far forward, sutured with fine silk. Then on the opposite side an ordinary submucous dissection is done. This can be done if there is a sufficient amount of cartilage behind that first removed, which is not too greatly bent to the side of main deflection. As the bend of septum passes from the anterior piece which lies obliquely across the right nares, let us say, to the left nose, there is often a small piece of cartilage, one-quarter to one-half inch in length, that remains fairly well set in the median line. In the first operation remove cartilage to the anterior end of this medianly set portion and begin on the other side behind this same part, so that we have a large

undisturbed portion of the cartilage of the septum left for support of the anterior end of the nose. This should prevent any later tendency of the tip from bending downwards, due to cicatricial contraction of the anterior operated area. As I have stated before, I have never seen this condition result, but believe the method just described a safe and conservative one. It gives a good result and saves tissue, and in all cases that should be our aim.

In one case the anterior inferior portion of the septum showed an old perforation from typhoid, about the size of a dime, with a marked left-sided deflection extending posteriorly, involving both bone and cartilage. The incision was placed sufficiently behind the perforation so that the opening in the septum was surrounded by cartilage. This was done to insure a firm anterior attachment for the freed mucoperichondrium, preventing consequently any tendency of the membranous septum from flapping upon blowing the nose. The greatest difficulty experienced in this case was due to the field of operation being set so far inside the nose, which necessitated considerable time in elevating the flaps, in order to keep the cartilage at the posterior portion of the perforation intact.

The cases described show to some extent the peculiarities met in this line of work, and also that they can be safely corrected and a good result obtained if sufficient care is exercised on the part of the operator.

XXXVIII.

THE OPERATIVE FINDINGS AND RESULTS IN MASTOIDITIS, ACUTE AND CHRONIC.

By J. M. Ingersoll, A. M., M. D.,

Cleveland.

It is the purpose of this paper to describe briefly the symptoms, operative findings and results in a few cases of mastoiditis. Naturally such a paper will contain little or nothing which is strikingly new, but the consideration of the symptoms and operative findings may help us to recognize similar cases early and by prompt operation prevent serious complications.

Case 1.—A boy, one year old. He had had a coryza followed by a purulent discharge from the right ear for five weeks. The temperature was normal, but the discharge from the ear was profuse, and the posterior wall of the auditory canal was bulging. On account of the profuse discharge and bulging canal wall, a mastoid operation was done and a large antrum filled with pus was opened. The discharge from the ear ceased in a few days, and the mastoid incision was entirely healed in two weeks. The hearing in this ear is now normal. This case illustrates the well-known fact that a profuse purulent discharge from the ear, continuing for more than four weeks, is an almost certain indication of infection of the mastoid antrum. If, in addition to the profuse discharge, there is some swelling of the posterior or superior membranous canal wall, the diagnosis of mastoiditis is positive, for the swelling is caused by the inflammation in the mastoid cells situated between the antrum and the auditory canal.

Case 2.—Male, thirty-five years old. Acute suppurative otitis for six weeks. Profuse purulent discharge with slight bulging of the posterior canal wall and tenderness over the tip of the mastoid. Temperature normal. Operation. The mastoid cells had practically all been destroyed, making one large cavity extending down to the tip, filled with granulation tissue

and pus. The whole tip of the mastoid was necrotic and was removed as a sequestrum, The recovery was uneventful and complete, with normal hearing. This case shows how rapidly the whole mastoid may become inflamed and even partially necrotic without causing very marked symptoms. This patient had remained at home only two days at the beginning of the acute infection in the ear, and had not consulted any physician until the day before the operation. The tenderness over the tip of the mastoid indicated an infection in the cells there, but one would naturally expect more evident indications of so extensive an involvement of the bone.

Case 3.—Male, twenty-one years old. Profuse purulent discharge from left ear for six weeks. Granulation tissue projecting through the perforation in the drum membrane. Some swelling and tenderness over the mastoid bone. Temperature 99.8°. Operation. All the mastoid cells were filled with pus and granulation tissue. The sigmoid sinus had been exposed by necrosis and was covered with granulations. There was a small extradural abscess just above the sinus. Recovery was complete in three weeks.

This case also shows how extensive the destruction of the mastoid may be without causing very decided symptoms. The mastoiditis was self-evident, but there were no symptoms indicating brain abscess or any involvement of the sinus.

Case 4.—Male, forty years old. Purulent discharge from the ear for seven weeks following an attack of tonsillitis. Temperature 99.6°. The whole membranous canal was inflamed and tender, and there was considerable swelling above and anterior to the ear, extending forward as far as the orbit. Posterior to the ear there was very little tenderness or swelling.

The discharge from the ear had been gradually decreasing, and it was thought that perhaps the swelling around the ear was due to the external otitis, so that the patient was kept under observation for a few days. Then tenderness over the antrum became manifest and the mastoid was opened. The mastoid cells extended well forward into the posterior root of the zygoma and posteriorly back and around the sinus. They were all filled with granulation tissue and pus, and the cells posterior to the sinus had broken down, causing an extradural abscess. The wound was packed wide open. On account of its size it filled in slowly, so that it was necessary to

dress it for eight weeks. It healed, however, without much depression and the hearing was normal.

In this case the swelling above and anterior to the ear and in the superior and anterior part of the canal wall was due to the infection in the cells which extended forward into the zygoma, and not to an external otitis. It would have been better to have operated when the patient was first seen.

Case 5.—Girl, three years old. She had had a severe coryza for four days, and was seen a few hours after she first began to complain of pain in the right ear. The drum membrane was red and bulging, and an incision was made in the posterior inferior quadrant. There was a free serous discharge which became purulent in two days. The temperature ranged between 101° and 103°. Twelve days after the incision in the drum membrane there was some swelling in the canal wall posteriorly, and the sternocleidomastoid muscle was slightly tense, with some fixation of the tissue over the tip of the mastoid. The latter two symptoms indicated that the cells in the tip of the mastoid were infected, and the mastoid was opened. The temperature came down to normal in three days, and for twelve days everything seemed to be progressing favorably. Then the temperature went up to 102° and a little granulation tissue appeared on the posterior canal wall. Under this tissue necrotic bone could be felt and a second operation was done at once. The posterior canal wall, down to the margin of the annulus tympanicus, the whole tip of the mastoid and part of the covering of the sinus, were removed, as there was some necrosis in all of these structures. Recovery after the second operation was complete in four weeks.

In this case the infection involved the mastoid, in spite of a free and early incision in the drum membrane, and although the first mastoid operation was as thorough and complete as the indications seemed to warrant, it became necessary to do a modified radical operation. The necrosis which developed after the first operation was probably due to the severity of the infection (streptococcus) and the low resistance of the patient.

Case 6.—Male, four years old. Chronic foul suppuration in left ear since infancy; middle ear filled with granulations. No tenderness nor swelling over the mastoid. Temperature normal.

Radical mastoid operation. A sequestrum containing most

of the mastoid cells and the outer covering of the sinus was removed. The roof of the middle ear and antrum was necrotic, exposing the dura, which was covered with granulations. Because the sinus and dura were exposed, the inferior part of the mastoid incision was left open and packed. The ear was dry and healing was complete at the end of two months.

In this case the chronic suppuration had exposed the dura and the sinus, and the wonder is that the child had not died of meningitis or sinus infection.

Case 7.—Boy, five years old. Profuse, foul, purulent discharge from left ear for fourteen months, following scarlet fever. Middle ear filled with granulations. Temperature 105°. Restlessness, nausea and semiconsciousness, made the diagnosis of meningitis almost certain and the prognosis very unfavorable.

A radical mastoid was done a few hours after the boy was admitted to the hospital. The roof of the middle ear and antrum was necrotic. The dura was inflamed and tense, and so it was uncovered over an area about three centimeters in diameter and incised. There was a little serous discharge through the incision. The day after the operation the boy was slightly better, but on the second day he was worse and died on the third day. No autopsy was permitted.

If this boy's ear had been operated upon before the meningeal infection had taken place his life would probably have been saved.

Case 8.—Female, thirty years old. Chronic bilateral suppurative otitis media. Facial paralysis on the left side, dizziness, nausea, headache and spontaneous nystagmus to the right. After turning to the left the nystagmus to the right was decidedly increased for forty-three seconds. Turning to the right, nystagmus to the left for twenty seconds. Caloric reaction present in the left ear, but sluggish. Hearing (acoumeter) left ear, one centimeter; right ear, eighteen centimeters. Fistula symptoms absent. Temperature 99.2°. From the above symptoms the diagnosis of paralabyrinthitis, with some involvement of the facial nerve, was made and a radical mastoid operation done. The sigmoid sinus was covered by such a thin bony cortex that it could be seen when the periostium had been removed. It was so far forward that it was necessary to expose the sinus in order to get working space.

In spite of the negative symptoms a careful search was made for a labyrinthine fistula, but none was found.

After the operation the facial paralysis improved, the spontaneous nystagmus decreased and the temperature went down to normal. On the tenth day the temperature went up to 102°, there was considerable headache and necrotic bone could be felt in the region of the horizontal semicircular canal. The mastoid incision was opened, and because a fistula in the horizontal canal was found, a labyrinth operation was done. There was some necrosis of the bone over the facial nerve, and a small sequestrum was removed, leaving the facial nerve exposed. At the end of three months this ear was dry, but there was still some facial paralysis.

The right mastoid now began to be sensitive, and the patient had some nausea and headache. There was a perforation in the posterior inferior part of the drum membrane, through which granulation tissue covering the promontory could be seen. The rest of the membrane was in fairly good condition, and there seemed to be no necrosis of the ossicles.

On account of the mastoid tenderness, headache and nausea, it seemed necessary to operate upon this ear also, in spite of the fact that the left ear was absolutely deaf and that the hearing in this ear might be made less acute. A modified radical mastoid was done. The mastoid antrum was filled by a cholesteatoma, but the rest of the mastoid appeared to be eburnized and was left untouched. At the end of seven weeks this ear was dry. It was very gratifying to have the hearing in this ear increase from eighteen to one hundred and ten centimeters (acoumeter).

In this case the facial nerve was involved before the operation, and the slight facial paralysis which remains cannot be attributed to the operation. Both the facial paralysis and the labyrinth infection would have been avoided by an earlier operation.

In nearly all of the above cases radiographs were taken of both ears, and in the cases in which only one mastoid was involved the normal ear furnished a standard for comparison. Invariably the diseased mastoid gave a dimmer picture than the normal one, the mastoid cells were indistinct, and the cell walls were partly or completely obliterated; whereas in the normal ear the cells were clear cut and distinct.

Stereoscopic radiographs of the head, taken to show the infected mastoid, were found to be exceedingly valuable helps in making the diagnosis and in determining the position of the sigmoid sinus, the relation of the mastoid cells to it, and how far forward the cells extended into the zygoma, and also the lower limit of the brain cavity over the middle ear and the antrum.

It is possible in many cases to make a positive diagnosis of exposure of the dura and sinus by studying the stereoscopic radiographs of the mastoid before such exposure has caused any clinical symptoms. In other words, stereopscopic radiographs often enable us to recognize the imminent danger of meningitis, sinus thrombosis and brain abscess before these infections actually develop, and by prompt operation to prevent their development.

In the acute cases the operative findings and the results obtained justify the following conclusions: A profuse purulent discharge from the ear continuing for more than six weeks is almost positive evidence of mastoid infection. If, in addition to this, there is some sagging of the posterior canal wall, the evidence of mastoid infection is certain. The sooner a mastoid operation is done the better is the prognosis for rapid recovery with normal hearing.

Elevation of the temperature, if present, helps to confirm the diagnosis; but a normal temperature does not speak against the diagnosis of mastoiditis.

Tenderness over the mastoid antrum or tip or over the root of the zygoma and fixation of the sternocleidomastoid muscles are all confirmatory signs.

In chronic suppurative otitis media, careful, persistent local treatment should be carried out for two months, if the condition improves during this time. If it does not improve in two months' time, or if there are any positive indications of involvement of the labyrinth, sinus or brain, then a radical mastoid operation should be done.

It is hardly necessary to state that these conclusions are not original with the author. They are the accepted teachings of all otologists.

XXXIX.

RADIOGRAPHY AS AN AID IN THE DIAGNOSIS OF MASTOID DISEASE.*

By Frederick M. Law, M. D.,

New York.

The radiograph alone cannot be depended upon in the diagnosis of mastoiditis, as there are various conditions which influence the appearance of the plate; but used in conjunction with other methods of diagnosis, it is an invaluable aid.

In order to interpret a plate correctly, one must be familiar with the clinical symptoms; therefore, when a patient is sent to the radiographer, a short note of the clinical findings should accompany the patient.

The normal mastoid is a pneumatic cavity containing bony partitions, hence there is very little resistance to the passage of the rays; the silver of the plate is easily affected and a dark area is produced; the bony partitions showing as light lines. The lateral sinus and emissary vein, being really large cavities, show as dark streaks in the bony structure of the skull, but where the sinus crosses the normal mastoid cells, the additional cavity of the sinus is merged with the cells and the outline is distinguished with difficulty.

A mastoid structure that is filled with pathologic material or covered with a hard cortex offers considerable resistance to the passage of the rays; the silver of the plate is not acted upon and an area lighter than normal is produced. In such a condition the lateral sinus, being a cavity, allows more of the rays to pass and shows as a dark streak through the light area.

This appearance of the lateral sinus in the mastoid structure is often a diagnostic point in determining the difference between a normal mastoid and one filled with fine granulations.

The auditory canal, if free from pus, shows as a dark spot, but if pus is present, the outline is made out with difficulty; then the location must be determined from the articulation of

*Read before the New York Academy of Medicine, Section on Otology, May 12, 1913.

the jaw, which is about one-quarter inch in front of the canal. The shadow of the pinna also aids in determining the position of the canal.

If the angle of the principal ray strikes the mastoid correctly, the entire mastoid region is shown clearly, and if the auditory canal happens to be straight and is perpendicular to the center of illumination, the small bones of the ear will stand out fairly sharp.

In the majority of cases the two mastoids are similar in structure; if there are large cells in one there will be large cells in the other. For this reason both mastoids are radiographed and the condition of the cells compared. The difference between the clear cells of the normal mastoid and the dull hazy cells of the diseased side is very plain.

The only conditions in which any doubt may arise is in the case of a double mastoid or an old chronic ear on both sides with an acute exacerbation in one. In these cases the cells of both sides are occluded and both plates show a hazy blur with the outline of the lateral sinus showing clearly. In such conditions dependence must be placed on the clinical symptoms, but if the plate is a good one, sclerosis may sometimes be determined by noting carefully the appearance of the shadow. The cells are completely destroyed and the mastoid resembles the bone of the skull; the skull and the mastoid seem to merge into one another and practically no outline of the mastoid can be seen, the anterior border of the lateral sinus standing out distinctly.

Mastoiditis is shown by the appearance of the cells as compared with the normal side. This may be from a slight hazy appearance to complete occlusion of the cells. In a mild condition there is a slight haze over the cells, but the bony partitions are still intact; as the condition progresses the haziness increases, light spots appear, which indicate collections of pus or spots of granulations. In a severe case the haziness becomes a dense white blur with no partitions visible, the lateral sinus standing out clear and the auditory canal barely showing. Cholesteatoma shows as a dark area in the midst of the white.

It is of course essential that the exposure of the two mastoids shall be similar, and the density and contrast alike, or there will be difficulty in comparing the two sides.

It is difficult to locate accurately the position of the lateral

sinus in a single plate made in the regular mastoid position, for the reason that some sinuses are placed deeper than others, and the deeper the sinus, the farther from the plate it is and the nearer the shadow is thrown to the auditory canal. Even so, the distortion will not be greater than one-eighth of an inch, and one can say the sinus is far forward or far back.

The location of the emissary vein is shown in the majority of cases.

A radiograph of the mastoid will show: The size and extent of the mastoid cells; the presence of pathologic material; sometimes the presence of sclerosis; the presence of cholesteatoma; the approximate size and position of the lateral sinus and emissary vein.

TECHNIC.

In making a radiograph the head is placed on a raised platform provided with a means for holding the head rigid; the ear to be radiographed is placed on the plate; the tube is centered so that the central ray will pass through the head to the mastoid on the opposite side on a line 15 degrees from the vertical and 15 degrees from the horizontal. This is an arbitrary rule which must be varied in some cases. With some patients the ear is placed higher than others, and in these the vertical angle can be lessened.

Some radiographers fold the pinna forward. If this is done the head is tilted slightly backward and the horizontal angle of the tube need not be so great.

A medium hard tube is used with sufficient exposure to produce good density and contrast.

XL.

A DISCUSSION OF THE MODUS OPERANDI OF GALVANIC NYSTAGMUS.*

By Lester Mead Hubby, M. D.,

New York.

Galvanism produces or affects nerve impulses through electrotonic currents or phases induced in the nerve. These cause phoretic changes in the tissues. Negative ions, such as oxygen, acids, etc., are drawn toward the anode; and positive ions, such as hydrogen, alkalies, etc., toward the cathode. Osmosis is altered, and may be reversed by this process during the passage of the current. Through the microscope a flow of fluid may be seen in a muscle fiber from the anode to the cathode, while the current is passing, with the production of a swelling at the cathode. ("Electricity," Chas. S. Potts.)

By these changes the nerve impulses, nerve irritability and conductivity are altered. In the anelectrotonic region the nerve irritability, conductivity, and strength of impulses are decreased; while in the catelectrotonic region the reverse is true.

Sufficient catelectrotonus of the normal vestibular nerve fibrils and terminations of one side of the head produces combined horizontal and rotatory nystagmus to that side of the head, whatever the position of the head. Sufficient anelectrotonus of the same results in nystagmus to the opposite side of the head.

Galvanism of both ears simultaneously with a double electrode causes no nystagmus unless one vestibular nerve, wholly or in part, is more sensitive than the other.

Catelectrotonus of one ear together with anelectrotonus of the other ear produces about double the amount of nystagmus, or results in nystagmus with about half the amount of amperage that is required in galvanism of one ear alone.

In destruction of the labyrinth of one side, the vestibular nerve, when still functionating, generally requires greater

*Read at the New York Academy of Medicine, April 11, 1913.

amperage to produce nystagmus than the nerve of the other side; and cathodal irritation of the normal side generally produces nystagmus with a slightly lower amperage than the anodal. This latter is explained by the presence of a more or less spontaneous nystagmus to that side.

The application of an electrode near the ear is at a great relative distance from the cristæ compared to the distances separating the latter, roughly speaking 5 cm. at least to less than 5 mm. respectively; and the diameter of the electrode generally used is greater than the greatest distance between the cristæ. Moreover, the galvanic current tends to diffuse in all directions from the point of application of the electrode.

Thus it is readily seen that all the vestibular nerve terminations are about equally affected in the ordinary method of testing.

Galvanic stimulation of individual cristæ or of the neighboring nerve fibrils is exceedingly difficult and in most cases impossible. It can only be done when the labyrinthine capsule is well exposed, as in a radical mastoid operation, and when the field can be made exceedingly dry, free from granulations, etc. I have succeeded in producing it in only one individual thus far. The cathode, an electrode about 1 mm. in diameter, was placed over the canals a few millimeters from their ampullæ seriatim. The different points of application were distant from each other about 5 to 6 mm.

As the cristæ are closer together than this, you can see how difficult it would be to sufficiently concentrate the current on one crista if applied near the cristæ themselves. By this method of application the current reaches a single crista, or at least the vestibular nerve branch going to the crista, in sufficiently concentrated power to produce a preponderating irritation of the nerve termination sought. (See chart No. 1.)

The galvanic current leaves a nerve in a condition of hyperirritability for a period lasting from a few seconds to fifteen minutes.

After the application of the anode the positive modification (Pflüger) lasts from a few seconds to several minutes.

There is a brief period of negative modification following cathodal irritation, followed by a positive modification lasting from one to fifteen minutes.

In positive modification the irritability of the nerve is increased, in negative modification, decreased.

This explains the fact that a doubtful spontaneous nystagmus is often increased to a demonstrable degree after galvanism of the ear.

In order to discuss the modus operandi of galvanic irritation of the vestibular nerves, it will now be necessary to consider the latter somewhat in a general way.

In the evolution and development of the vestibular nerve system, stimuli induced by head movements and changes in intralabyrinthine pressure were the only factors. The automatic interpretation of these impulses in the cerebellar and paracerebellar nuclei was always in terms of relative position and changing positions of the head.

Unnatural, unaccustomed stimuli, such as the application of cold or heat, or of the galvanic current, to a small area near the static labyrinth, inducing centripetal vestibular nerve impulses, could, therefore, only be interpreted as relative position and changing positions of the head. The same can be said of sufficient changes in intralabyrinthine pressure, however induced.

Conjugate deviation of the eyes (the slow component of nystagmus) and somatic muscular movements away from the canal stimulated and in the plane of that canal, are the automatic responses in all probability of the cerebellar cortex to such stimuli. For Bárány has demonstrated many of the reaction centers in this part of the cerebellum, and the slow component of the nystagmus is merely the reaction movement of the eyes. The rapid component of the nystagmus is the reflex central correction of the conjugate deviation of the eyes, and its centers are probably located in the posterior portions of the median frontal convolutions of the cerebral cortex, the location of the centers for voluntary movements of the eyes.

Nystagmus production is the result of the action of a long chain of neurons, and is not to be compared with the action of a single neuron, such as that of a motor nerve and its muscle. To remind you how long this chain is I have drawn a chart showing the probable connections, omitting the cerebral connections. You will see that at least twenty-three neurons are involved in the production of vestibular nystagmus, without counting the cerebral connections. (See chart No. 2.)

The combined centripetal impulses from the cristæ of both sides, under normal conditions of body rest, produce a certain

condition or tonus of rest or equilibrium—a nystagmus equilibrium. If the impulses from one or more cristæ of one side are increased or decreased, relatively or absolutely, to a certain degree, this equilibrium is upset and nystagmus ensues until the equilibrium is reestablished.

This explains the long continued nystagmus following destruction of one static labyrinth.

In man the end organs of the vestibular nerves in the ampullæ of the semicircular canals are situated in the row of cells (sustentacular cells) with their hair-like prolongations into the hat-shaped gelatinous cupulæ. The hair cells are most numerous near the summits of the cristæ.

There are the following possible ways in which these nerve terminations can be irritated, namely—by variations in pressure, or stretching, or torsion, or combinations of any or all of these forces.

That variations in pressure must be a factor is evident from the following considerations:

1. Nystagmus occurs when there are sufficient changes in intralabyrinthine pressure, such as is produced in slight effusions of serum, pus or blood, in the static labyrinth, etc.

2. Ewald's experiment is not a simple production of endolymph movement, but with the latter there must be changes of endolymphatic pressure. For the membranous labyrinth is a closed tube, the endolymph is practically incompressible, the walls of the membranous labyrinth are surrounded by the practically incompressible perilymph, and the saccus endolymphaticus within the dura by the equally incompressible cerebrospinal fluid in the cranium.

Note.—Possibly the saccus endolymphaticus has the function of ameliorating great changes in pressure in the membranous labyrinth by its own expansion or shrinkage, acting thus as an equalizer of pressure.

Therefore, on forcing a piston into or against one membranous canal, the endolymphatic pressure must be raised, temporarily at least; likewise on suction the endolymphatic pressure must be lowered.

3. Some animal cristæ are apparently arranged so that they seem capable of responding only to changes in pressure. (Siredon Mexicanus, etc.)

It is not within the scope of this discussion to attempt to explain why pressure on the horizontal canal toward the am-

pulla has the reverse effect of that on the vertical canals. Suffice it to say that it is only necesasry to assume that the anatomic and histologic arrangement of the cristæ is such that movements of the endolymph on rotation of the head produce greater irritation in one direction than in the other, in the different canals.

Above normal irritation of any crista, whether induced by quick head movements, galvanism, rotation, disease, pressure, or caloric means, causes nystagmus to that side of the head.

On the other hand, if through any of the above agencies any crista is put in the condition of subnormal irritation, then nystagmus results to the opposite side of the head, not alone through overaction of the opposite side, but also through hypostimulation of the same side.

There must then be an average condition of rest, a nerve impulse tonus, if you will, of these nerve terminations in each crista, as suggested by Goltz for pressure only. For instance, each crista must be continuously transmitting impulses concerning the exact status of pressure, torsion, stretching, etc.

It is like a man wearing a hat—the weight of the hat and the compression of the hat band produce nerve impulses continuously to the brain. These impulses are soon disregarded. But if there is a sudden change, such as a gust of wind tugging at the hat, the nerve impulses are greatly changed, and more or less automatic reflexes result, such as grasping at the hat, etc.

So with the crista—its automatic responses are nystagmus and somatic muscular movements.

A certain amount of increase of nerve impulse tonus of any crista produces nystagmus toward that side, and somatic muscular movements away from the canal and in the plane of the canal; a certain amount of decrease of the nerve impulse tonus of a crista will produce nystagmus away from the canal affected, and somatic muscular movements toward and in the plane of the canal.

If we call irritants only those forces which increase the nerve impulse tonus of the cristæ of one side of the head, then we see that when they are exerted against one side of the head they cause conjugate movement of the eyes and somatic muscular movements away from the irritated side.

On the other hand, if depressants are applied to the tonus of the cristæ of one side of the head, the reverse occurs, name-

ly, conjugate deviation of the eyes and somatic muscular movements toward the affected side.

Let us now consider why it is that in irritants such as disease, head movements, galvanism, etc., applied to all the canals of one side, the result is always a combined horizontal and rotatory nystagmus to that side.

If you will examine this model of the three canals, remembering that hyperirritation of one crista produces nystagmus toward that side of the head and in the plane of that canal, you will find the following:

Irritation of the right horizontal canal will produce horizontal nystagmus to that side; that of the superior canal will produce a rotary counter-clockwise movement of the eyes forward and to that side, which viewed from the front is rotatory to that side; irritation of the posterior canal will produce a rotary counter-clockwise nystagmus backward and to that side, which viewed from the front is rotatory to that side. Therefore, the resultant of these three must be a combined horizontal and rotatory nystagmus to the side irritated.

Bárány's explanation of galvanic nystagmus rests on the theory that there is a nystagmus equilibrium, and that there is altered conductivity of nerves in electrotonus. He claims that the slightest change in the nerve impulses from either side of the head upsets this equilibrium and results in nystagmus, the direction of the nystagmus being toward the side which is relatively stimulated. In catelectrotonus the conductivity of the vestibular nerve is increased. Therefore, nystagmus results toward that side on account of the increased transmission of the nerve impulse. In anelectrotonus the conductivity of the vestibular nerve is decreased. Therefore, the impulses are more or less blocked on that side, and those of the other side are relatively increased, and nystagmus results to the opposite side.

As pointed out by Brüning, if this theory were true, on the production of cold caloric nystagmus to the opposite side, the application of the cathode to the irritated side should increase the nystagmus to the opposite side, as the nerve conductivity is increased in catelectrotonus.

Instead of this, the nystagmus is stopped by the catelectrotonic effect on the nerve.

The same is true of heat caloric nystagmus to the same side

being overcome in the production of anelectrotonus of that side.

Brüning's theory rests on the physical movement of matter in the neighborhood of the cristæ toward the cathode and away from the anode.

It would be difficult to harmonize this with the fact that nystagmus of the same kind and degree is produced, whether the electrotonus occurs near the cristæ or in the nerve itself when the cristæ have been destroyed.

With our present limited knowledge, the following theory seems to explain galvanic nystagmus in health and disease of the vestibular neurons:

1. Each crista with its hair-like processes is continuously transmitting afferent impulses of its condition in respect to pressure, stretching, or torsion, or combinations of these three. The condition of the nerve terminations in the crista, hair cells, etc., is then either in a normal state of irritation, a hyperirritated condition, or a hypoirritated condition.

2. The sum of these impulses from all the cristæ in body rest and under normal conditions, produces from the centers efferent impulses to the eyes, resulting in a nystagmus equilibrium.

3. Sufficient change in the relative amounts of these impulses upsets the nystagmus equilibrium and nystagmus results.

4. Impulses from any crista on one side of the head, however induced, indicating sufficient increase of irritation, cause nystagmus to that side of the head; those showing a sufficient decrease, cause nystagmus to the opposite side of the head.

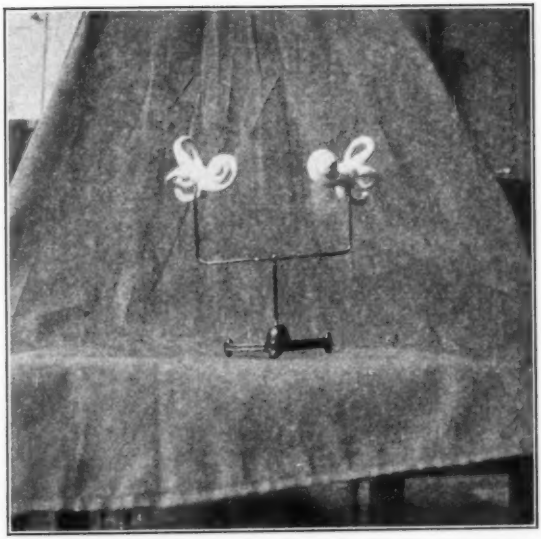
5. The nerve impulses from the vestibular nerve and its terminations are altered during the production of electrotonus.

(a) Catelectrotonus induces a hyperirritated condition of the nerve and nerve terminations, an increase of afferent impulses; therefore, a nystagmus toward that side of the head.

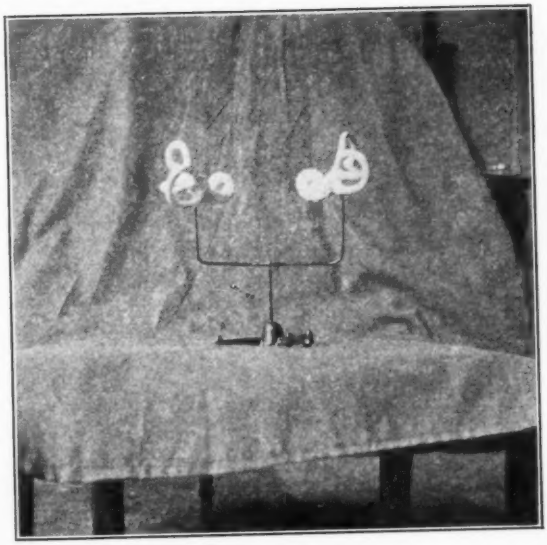
(b) Anelectrotonus induces a hypoirritated condition of the nerve and nerve terminations, a decrease of afferent impulses; therefore, a nystagmus to the opposite side of the head.

6. The character of the nystagmus will vary with the relative amount of irritation of the different nerve terminations, cristæ, etc., and will be a resultant of the same. For instance, if the crista of the horizontal canal is mainly irritated, the nystagmus will approach the horizontal type.

Anterior View of Model.



Anterior View of Model.



Posterior View of Model.

XLI.

EXHIBITION OF RADIOGRAMS ILLUSTRATING THE PATHOLOGY OF MASTOIDITIS.†

By Henry S. Birkett, M. D.,

Montreal.

I wish to express to you my appreciation of the honor of the invitation extended to me to take part in this evening's discussion, but much regret that owing to a change from the original date to the present, it conflicts with the conduct of my examinations in the University, hence my reason for being absent.

When reflecting upon the value of radiography in diseases of the frontal sinuses and antra, it occurred to me that perhaps the same method might be applied to the mastoid process, and thus, independently of any work that had been done elsewhere, I had my first plate made in 1908. It was taken with the patient lying on his face, and the result was not satisfactory, as only the cells and the tip of the mastoid were visible. Studying the matter still further, I learned that Schier of Breslau had done some work along these lines, but his plates were taken with the patient lying on his side. In 1909 I again took up the work, having my plates taken with the patient lying on his side. For a description of the technic I am indebted to Dr. A. Howard Pirie in his article on "Radiography in the Diagnosis of Mastoid Disease,"* which is as follows:

"The patient should lie prone on a firm couch. The head is supported on an inclined plane making an angle of twenty-five degrees with the plane of the couch. The photographic plate rests on this inclined plane. The head is rotated 90 degrees, so that the patient looks directly to his side: this brings the mastoid into contact with the plate. The pinna of

†Presented before the New York Academy of Medicine, Section on Otology, May 12, 1913.

*Reprinted from "Archives of the Roentgen Rays," September, 1912.

the ear is turned forward so as to obscure the mastoid as little as possible. The source of X-rays is placed vertically above the head, and the perpendicular ray is made to fall on a point two inches above the highest point of the pinna. The mastoid on each side must be skiagraphed separately. The glass of the focus-tube should be nine inches away from the hair. The exposure required will turn a Sabouraud pastille, placed at 2 centimeters from the glass, to one-third of the B tint. A medium hard focus-tube (4 to 5 Benoist) with 30 milliamperes for fifteen seconds from a Snook apparatus, gives a plate which should be fully developed in seven minutes (Ilford plate and developer). Skiagraphs of both right and left mastoids must be made of every case, as a single skiagraph of one mastoid is of little value. A different focus-tube should be used for each mastoid, as it is rarely possible to get one tube to remain constant in vacuum for both exposures. Both focus-tubes must, of course, be of the same hardness. This is one of the most important points in the technic, viz., to have two similar tubes of equal hardness and quality. The American made tubes lend themselves to this better than any others I have used."

The value of radiography in affections of the mastoid process is to demonstrate first of all the anatomic characters of the mastoid process itself, diploetic, pneumatic or sclerosed. It further gives us in many cases the position of the antrum and the position of the lateral sinus in its relation to the mastoid process, and in many, as shown in some of the plates, the emissary vein is distinctly seen.

Clinically, we are enabled by radiography to recognize when the mastoid process has become involved in acute inflammatory or purulent infection, by a distinct blurring or veiling of the cells. Should the disease have existed for some time, leading to destruction of the cells, this is often indicated by distinctly enlarged cavities, which occurring over the lateral sinus in some cases shows it definitely exposed as a dark area. One case I have on record—unfortunately the plate is not on exhibition owing to its having been broken—distinctly shows a sinus thrombus. This was evidently due to the fact that the thrombus was not only surrounded but infected with pus.

I wish, however, to make it distinctly clear that I am not an advocate of radiography as applied to diagnosing diseases of

the mastoid process exclusive of one's clinical experience; I only look upon it as one of the nice adjuncts to one's work and I do not allow my judgment to be biased by any findings of the radiogram. But I will say this, that my readings of the radiograms have in operative cases always been confirmed.

I wish to acknowledge the valuable assistance of Mr. McNeil of the X-ray department of the Royal Victoria Hospital in the development of this work from its very beginning.

XLII.

FURTHER OBSERVATIONS ON THE TECHNIC IN THE AFTER-CARE OF THE RADICAL MASTOID OPERATION.

By Philip Hammond, M. D.,

Boston.

To achieve progress in any endeavor it is necessary to keep constantly in mind the goal toward which we are striving. Under no condition should we allow our efforts to be diverted from the main object by attempts, however zealous, to prove the truth of preconceived notions, unless those notions lead in the most direct way to the solution of the questions under consideration.

Pedantic as this may seem, it strikes directly at the cause of many exploited "methods"; for often one who has been instrumental in the development of an idea, or of some operative procedure, will become so fixed in his habits that he will tolerate no change in his accustomed routine, even though inconceivably superior methods have been adopted by his coworkers.

The advisability, nay even the necessity, of constantly changing one's mental attitude toward the treatment of the radically operated mastoid has been impressed upon the writer during an experience covering practically twenty years; if we but analyze the progress that has been made in this time, we shall see that it has come about by attempts to accomplish a definite result in the simplest possible way.

Even with the first cases it was recognized that success could not be attained without epidermization of the cavity, and to this end a postauricular opening was left through which various cutaneous flaps were introduced to aid the epidermis from the meatus in spreading throughout the inside of the bone.

Following the tendency of that time, the wounds were

packed almost daily with gauze, and needless to say the granulations thrived upon this treatment. Then to aid in the process of epidermization, small Thiersch grafts or islands of epidermis were introduced. The permanent opening behind the ear proved so disfiguring that it was soon abandoned.

Opinions still vary as to certain details of the complete radical, which will be taken up later, but authorities are agreed up to a certain point. It is generally conceded that the bone operation should be most thoroughly and painstakingly performed, all crevices smoothed out, and all projections removed. Some form of plastic operation is then performed upon the posterior part of the cartilaginous meatus, to serve the double purpose of free access to the cavity for inspection and treatment and to furnish a dermal covering for a part at least of the mastoid. The type of this operation varies with the demands of the case and the habits of the operator, but the tendency is to obtain a meatus that will be sufficiently large to readily allow of all after-treatment of the entire cavity. The posterior wound is now generally sutured throughout, with the object of obtaining healing by first intention. The method of suturing that has given by far the best results in the hands of the writer is by means of the small metal clamp, or Michel suture. Not only is the apposition much better than that generally obtained, but they can be applied much more rapidly than ordinary sutures. Only one failure has been noted in an extended series, and that was in a case that had been subjected to repeated operations. With the object of preventing hemorrhage from the incision, a dilute solution of adrenalin chlorid was injected subcutaneously over the operative field as a preliminary, after the method of Jansen. There was a general breaking down of the wound and some sloughing; but this could not properly be attributed to the method of suture.

From this point on we encounter the greatest diversity of opinion as to the proper course to follow. Roughly speaking, the exponents of the various methods may be divided into two classes: those who use the skin graft to aid nature, and those who do not.

Before attempting to take any stand on the subject we should remember, first, that our whole aim is to favor the complete epidermization of the cavity, and secondly, that this

should be accomplished as speedily and with as little distress to the patient as possible. The question then rapidly narrows down to one of results. Which method, or combination of methods, is most likely to show a favorable outcome? In this query we are likely to be led astray by prejudice, or by the fact that familiarity with a certain routine may have made the operator an expert with that particular method. Under these conditions any observer can scarcely avoid a bias toward the procedure which produces the best results at his hands, although the skill with which he administers his technic may be more of a factor in the successful issue than the means adopted.

To counteract this tendency, and to overcome as far as possible the objections which necessarily obtain where the operative work is carried on at the hands of one man, some of my colleagues at the Massachusetts Charitable Eye and Ear Infirmary have joined with me in an attempt to search out the truth of this matter. Cases have been operated upon by all the methods and the results noted. In addition to this, as many as possible of the cases operated upon for several years past have been requested to report at the hospital for observation. These cases have been carefully examined and the results freely discussed by advocates of the different methods of operation; so that the cases have been subjected to the criticism of the opponents as well as of the partisans of the methods employed. As a result of this inquiry several facts seem to have been established: first, the end results do not appear to favor any particular procedure, although the length of treatment was lessened in the cases subjected to grafting. Second, the cases in which the primary graft was used apparently did as well as those in which it was applied at a later stage. Third, as was to be expected, the best results were observed in cases where the most careful bone work had been done.

One of the interesting observations arising from a study of this series was the comparison of the very thorough exenteration practiced today with that done several years ago; the most recent cases allow of much better access to the middle ear, and the complete removal of the facial ridge leaves an extremely smooth cavity. This naturally aided in the healing of the wound, particularly where skin grafts were used. Many of the older cases had prominent ridges left at the site of the posterior canal wall, not only greatly interfering with the inspection of the ear, but actually handicapping the recovery.

Another point on which improvement has recently been made is in adopting some device to keep the meatus permanently enlarged. In a majority of the older cases it had narrowed down so that it did not provide room for thorough inspection of the cavity. In the hands of the writer the best results have been obtained by the use of a Koerner flap. This is carried well into the concha, the base of the flap being made as wide as possible. The resulting tongue is then pulled forward out of the meatus, and the skin of the flap separated from the cartilage, much as a submucous operation is done in the nose. The cartilage is now removed from the flap to its base. A deep buried chromicized gut suture is then passed through the base of the flap from behind and caught into the periosteum of the posterior edge of the wound, thus holding the meatus well open. To overcome the rather pointed corners in this artificial meatus, a short piece of large caliber rubber tubing is slit lengthwise, so that it is in cross-section like the letter C and can be expanded to fit any size canal. This is placed just at the entrance of the meatus and completely filled with gauze, continuous with the packing of the wound. It serves the double purpose of giving a better contour to the meatal edge, and of preventing the packing becoming so adherent to the edge of the wound.

While in the exceptional case very nearly if not quite as rapid healing takes place in the non skin-grafted cavity, this result is by no means uniform. With a reasonably small cavity, and flaps so placed from the canal as to advantageously assist in the spread of new epidermis, a favorable result is almost assured without further assistance. This is quite in accord with the teachings of general surgery; but one must not expect the skin to bridge a gap of more than half an inch.

With a large cavity it would seem to be only reasonable to expect that a graft might render valuable assistance in shortening the treatment. Indeed, the brilliant results which followed the use of some of the first Thiersch grafts for this purpose led many operators to adopt this method in all cases. Some always apply the graft at once, others as invariably await a later stage, commonly from the fourth to the tenth day. One operator advises further curettement of the cavity at this time, as a more certain means of encouraging the graft to take.

That some, at least, of this abundance of detail is needless, is attested by the results in certain cases, which from neglect have disappeared from observation almost immediately after operation, and without skin graft, only to reappear after months or years with a completely epidermatized cavity.

It is thought by many, however, that a suitable graft, successfully placed, lessens the period of convalescence decidedly. The chief objection advanced against its employment seems to rest on the fact that some operators experience a difficulty in having it "take."

Due care in the preparation of the cavity, however, will materially aid in accomplishing a successful result. If, under the best of conditions failures occur, something is wrong with the technic. In this the writer speaks not alone from his own experience, but from that of his colleagues as well. In the hands of several operators at our hospital the results are practically uniform.

No attempt is made at the present time to apply separate small grafts. A large thin graft, two inches or more square, is cut from the thigh with an ordinary amputating knife. The skin is immediately spread out on the place from which it was cut, exactly as if it had not been severed from its attachments. A square of crepe de lise is then laid on it, and the skin and crepe together removed from the thigh. This simple device absolutely prevents rolling or curling of the edges. A piece of ordinary gauze—or rather, several layers which have previously been rolled into a cone are then placed under one corner of the crepe and skin, in such a manner that the entire apex of the cone and at least one side is completely covered with skin, the raw side out. One side of the cone is purposely left free of skin, as this side goes into apposition with the anterior wall of the canal, which is already covered with epidermis. The cone, or plug, is then introduced through the meatus, the point guided toward the middle ear, and then the gauze of the plug gradually fed into the cavity, expanding and completely filling it.

It must be remembered that the graft is adherent to a thin, smooth, silky crepe, and the pressure of the mass of gauze causes this to reach every part of the cavity, even where the meatus is much smaller than the inside, the gauze expanding in much the way that a bristle probang does. This gauze is

allowed to remain undisturbed three or four days, at the end of which it is gently removed. The crepe de lise comes with it, but the skin never. In the interval it has become firmly attached to all denuded surface. There are naturally some loose ends where the graft is superimposed upon the epidermis of the meatus, and there are necessarily reduplications or folds in the graft, as it is impossible to adapt a flat surface to the inside of a sphere without such. These loose pieces finally slough out, and during the process there is a very active suppuration to deal with. In most of the cases the superficial layers of graft are cast off, but underneath there is the firm pink appearance of new skin, rapidly paling out. Immediately following the removal of the gauze plug it is possible to demonstrate that the graft has "taken" by pulling upon it. It stands a very considerable strain, and if we succeed in detaching a piece, it causes bleeding, showing that it is definitely attached to its new resting place.

This method of applying the graft was described by the writer several years ago, and has been in use continuously since. It appears to be simpler in execution than the Ballance method, and quite as certain of a successful result. The graft may be applied either at the primary operation or at a later period.

Following the throwing off of the superficial layers of epithelium a simple cleansing of the cavity with hydrogen peroxid may be permitted, but aside from keeping out the products of suppuration, the less treatment given the better. The writer does not favor the use of drying powders, scarlet salve or other medications, as they are apt to produce crusting and deceive one as to the true condition of the cavity. Under no conditions should packing of the cavity be continued.

Cases where the skin does not "take" pretty generally throughout the cavity are rare; but if such occur, and granulations form, they should be kept down by the free use of caustics. This is substantially the treatment that must be followed if no skin graft be used.

In summing up the record of progress, emphasis should be laid on the fact that while once the radical mastoid operation was looked upon as too severe a procedure to which to submit our private patients, it is now so treated that recovery takes place in as short a space of time as in the acute mastoid; and

that while once the dressings were painful, protracted, and called for the exercise of as much skill as the operation itself, now, thanks to our modified technic, they are practically painless, and every contingency has been so carefully guarded against in the operation that only a minimum amount of after-care is required.

REFERENCES.

Ballance: Transactions Ninth International Otological Congress, 1912.

Freeman: Skin Grafting, 1912.

Hammond: Journal American Medical Association, November 17 1906.

XLIII.

THE FAUCIAL TONSILS AS A FOCUS FOR SYSTEMIC INFECTION.*

By George E. Shambaugh, M. D.,

Chicago.

The close relation between acute rheumatic fever and sore throat has long been recognized. Formerly it was often assumed that the sore throat occurring in connection with acute rheumatism was but a local manifestation in the pharynx of the general systemic condition. It is now generally believed, however, that the sore throat in these cases represents the focus of entrance for the systemic infection. Furthermore, it is now quite generally recognized that sore throat, for the most part acute tonsillitis, is very often the immediate cause for other systemic infections such as acute endocarditis and acute nephritis. It has not been so generally appreciated that the faucial tonsils are very frequently the focus for chronic systemic infections such as chronic arthritis, commonly known as arthritis deformans, chronic neuritis, cardiovascular degeneration and chronic nephritis.

In the *Archives of Internal Medicine*, April, 1912, Dr. Frank Billings, of Chicago, discusses the subject of "Chronic Focal Infections and their Etiologic Relations to Arthritis and Nephritis." This work was done on patients in the Presbyterian Hospital of our city, and it has been my privilege to examine the throats of many of them and to enucleate the faucial tonsils in certain of the cases where these structures were suspected of harboring foci for the systemic infection. The observations on these cases, and on a large number of other cases of focal systemic infection referred to me during the past few years, have impressed on me certain facts. First, as regards the relation of the faucial tonsils to systemic infection; second, as regards the conditions found in the tonsils in

*Read before the American Laryngological Association, Washington, D. C., May, 1913

cases where these structures were clearly shown to harbor foci for systemic infection, and third, as regards the proper treatment for tonsils suspected of containing foci producing systemic infection. In this paper I wish to discuss briefly some of the conclusions which I have reached regarding these questions.

In the first place, I believe that the general practitioner, as well as the specialist, has not, as a rule, fully appreciated the importance of the relation existing between infections, acute as well as latent, of the faucial tonsils and certain systemic conditions. I believe that in general the importance of this relation has been more fully appreciated by the leading internists than it has by the specialists. This is, perhaps, to be expected, because the consulting internist is the one to whom such cases are referred. The internist is, moreover, familiar with systemic conditions caused by other foci of infection, such as abscess of the gums, chronic appendicitis, infection of the genital tract, cholecystitis, etc. The internist is, therefore, prepared to recognize in a structure like the faucial tonsil, which is so frequently the seat of violent acute streptococcus infection, not only the source for acute systemic infection, such as acute rheumatic fever, acute endocarditis and nephritis, but also the seat of persistent latent chronic foci of infection capable of causing chronic arthritis, chronic neuritis, cardiovascular degenerations, nephritis, etc. The attention of the specialist, on the other hand, is naturally centered more on the purely local conditions. He cannot be expected to have that fuller appreciation of the systemic conditions which experience brings to the internist. It is so easy for the specialist to conclude that because he has not in his experience recognized systemic conditions resulting from tonsil infection, therefore these systemic conditions do not result from disease of the tonsils. No greater mistake in reasoning than this can be made. It would be just as logical for an otologist to assert that because he has never seen a brain abscess develop in any of his cases of purulent otitis media, therefore purulent otitis media is not the chief cause of brain abscess. Dr. Billings (l. c.) states: "There can be no other reason for the prevalence of rheumatism found in childhood than the frequency of local infections in the throat and nose. Quite as frequently children have endocarditis without other symptoms

of rheumatic infection, which has its source in the throat. I think anyone who has noted the difference in the physical and mental make-up of a child before and after the removal of large amounts of lymphoid tissue in the throat, must be convinced that the change is due not only to a better air space, but also probably to the removal of a constant, even though it may be slight, systemic infection."

As regards the appearance of the faucial tonsil which is a focus for systemic infection, it is quite clear that the size of the tonsil is no index of the menace this structure may be to the individual. A large tonsil is often chronically diseased or the seat of repeated violent acute infections and, therefore, a constant menace as a focus for systemic infection. On the other hand, one sees a great many very large tonsils where the whole appearance is one of simple hypertrophy, and where there is no history of attacks of acute tonsillitis. The danger from tonsils of this type is, as a rule, very slight when compared with the danger from a small tonsil which is subject to repeated acute infections. At the same time the children with these enormous, apparently harmless, faucial tonsils often do appear illnourished and generally below par. Very frequently, too, in such cases the removal of the tonsils results in such an immediate and astonishing improvement in general health that it can hardly be accounted for except on the assumption that these enlarged, harmless appearing tonsils often contain foci for a mild systemic infection. The enlarged tonsil in the adult, which I have come to regard with suspicion as the possible focus for chronic systemic infection, is often more or less edematous and congested. The surface is more irregular, caused at times by deep ragged fissures, at other times by swelling of the parenchyma between the meshes of hypertrophic connective tissue stroma. In many of the tonsils there are dilated crypts containing the characteristic foul smelling, cheesy deposits which in many cases from time to time produce circumscribed acute infection, as shown by the local congestion and soreness. At such times the patient experiences more or less general malaise and dullness. Not infrequently one can, by pressing on the base of a tonsil, express from the depth a creamy fluid exudate which is largely pus, often filled with virulent streptococci. These enlarged tonsils in the adult are frequently quite fibrous and hard, but in some the swelling is

largely of the parenchyma and the tonsils remain soft and compressible.

Another suspicious type of tonsil frequently met with in adults is the very small fibrous tonsil. This condition is found in individuals who, for many years, have been subject to a great many attacks of acute tonsillitis. I have several times removed tonsils which were so shrunken as to represent scarcely more than a thin band of fibrous scar tissue, from which the macroscopic evidence of parenchyma had been largely eliminated and where the structure was so firmly adherent to the underlying tissue that it could not be pulled out of its bed, as is done before dissecting the tonsil free from the pillars. The patient is often not aware that the recurring attacks of sore throat are in reality recurring attacks of acute infection in the faucial tonsils. It is only by seeing the patient during an attack of sore throat that the real character of the trouble can be diagnosed. The amount of local discomfort, as well as the general symptoms, fever, headache, etc., are, as a rule, not very severe as compared with attacks of acute tonsil infection occurring in an enlarged faucial tonsil. I have seen a number of cases of chronic arthritis and chronic neuritis occurring in patients who had for years suffered from this type of tonsil trouble, where each fresh attack of tonsillitis was followed in a few days by an acute exacerbation of the chronic systemic infection, and where the enucleation of the small insignificant looking fibrous tonsils resulted in a complete cessation of attacks of acute sore throat as well as a disappearance of the chronic systemic infection and a remarkable improvement in the patient's general health.

Everyone is familiar with the small tonsil embedded under a fold from the anterior pillar and the tonsil with a deep horizontal fissure separating the upper from the middle thirds, and with the fact that such tonsils are unusually susceptible to acute infections which, therefore, especially predispose them to the development of latent foci capable of causing systemic infection.

There is another type of faucial tonsil that I have learned to regard as a frequent source for systemic infection. This is the tonsil stub remaining after partial removal or where the tonsil has been subjected to ignipuncture or surface cauterization. In these cases the surface scar seems often to

predispose to the retention of infection in the pockets of the remaining stub, causing repeated attacks of acute tonsillitis as well as systemic infection.

Occasionally one observes near the surface of the tonsils a sealed pocket filled with yellow pus. The tissue immediately surrounding such a pocket of pus often shows from time to time an acute reaction with swelling and congestion. On the other hand, such pockets are often observed when there is no evidence of an acute condition in the tonsils which would suggest tonsil infection. Pockets of this sort in the faucial tonsils frequently constitute the foci for chronic systemic infection. The size of the pocket of pus is not an important factor. Small foci seem to be as dangerous as a larger abscess. It is often overlooked that such foci may develop in the depth of a tonsil. As a matter of fact, one discovers such foci much more frequently near the bottom of the tonsil than near the surface. It would seem that the foci developing near the surface of the tonsil find an opening and heal more readily than when they develop near the bottom of the crypts. The presence of latent foci can sometimes be suspected from the appearanec of the tonsil as described above. On the other hand, I have often been surprised after removal of a tonsil suspected of causing systemic infection to discover near the base a pocket of pus, when neither the surface appearance of the tonsil nor the history suggested tonsil infection. Such experiences have taught me not to be too dogmatic in asserting that a tonsil may not be the focus for systemic infection simply because there is nothing suspicious in its appearance.

In this connection I wish to call attention to a condition which may occasionally follow what seems to be a complete enucleation of the tonsil. This is the formation, in the process of healing and contraction, of a small fistula which later may become the seat of an acute purulent infection with a circumscribed abscess formation in the bottom of the fistula. This condition I have seen in three cases. In two of the cases the abscess was located about the middle of the tonsil region, and the acute reaction occurred more than a year after the tonsil enucleation. In the third case the fistula was observed three months after the tonsil operation. There had been no acute symptoms, and the fistula led to a small cyst the

size of a pea, filled with pus containing staphylococcus, and located in the region of the supratonsillar fossa.

Chronic foci of infection in the faucial tonsil, just as such foci occurring elsewhere, represent a condition which has had its origin in some acute infection. In most of the cases the patient gives a history of such acute infections, consisting in attacks of acute tonsillitis. In many of the cases with chronic foci in the tonsils there may not have been an acute attack of tonsillitis for a number of years. In all cases of chronic systemic focal infection, where the competent internist is unable to discover evidence of foci elsewhere, the faucial tonsil should be suspected. Usually when the tonsils are at fault there will be a history of attacks of sore throat, tonsillitis, and a careful examination of the tonsils will disclose evidences of chronic infection.

As regards the proper treatment of faucial tonsils suspected of harboring foci giving rise to systemic infection, this should be the same as the treatment of such foci of infection occurring elsewhere in the body. The treatment consists in the thorough removal of the suspected foci. In the case of the faucial tonsils, I know of but one way by which this result can be assured. This consists in the thorough enucleation of the faucial tonsils. Whatever theoretical arguments may be urged against tonsillectomy as a routine procedure, when operating on enlarged faucial tonsils in children, such arguments should not prevent us from the thorough enucleation of faucial tonsils, whether they be large or small, when these structures are suspected of harboring foci causing systemic infection. Only in those cases, especially in adults, where the danger of a serious bleeding is increased because of a high blood pressure or a slow coagulation time, should the enucleation of the tonsil give place for less radical measures. Here we must be content with efforts to cleanse the tonsil pockets and possibly also with cautiously slitting them open. Surface cauterization or ignipuncture should never be resorted to, as these only tend to increase the trouble.

The enucleation of the faucial tonsils in children requires a general anesthesia. In adults I prefer, as a rule, to do the operation under local anesthesia, for the following reasons: The operation can be done with the patient in the upright position; the annoyance from bleeding is very much less; the

operation can be done with less traumatism to the surrounding structures, and the discomfort from soreness is less than where general anesthesia is employed. All cases are operated at the hospital. The patient remains at the hospital over night, or for several days if he wishes. I begin usually about 2 p. m. When local anesthesia is to be used the patient goes without lunch in order to decrease the discomfort from gagging while working in the throat. A hypodermic containing one-sixth grain of morphin with atropin is given just before the patient is brought up to the operating room. I use a 5 per cent solution of cocain in 1/1000 adrenalin, rubbed over the attachment of the tonsil. A one-half per cent solution of novocain, to which a few drops of adrenalin are added, is freely injected under the mucous membrane along the margin of attachment on the tonsil on both the anterior and posterior pillars. A deep injection of this solution is made in the lymphoid tissue at the lower part of the tonsil, and another is made in the base of the tonsil directly through the anterior surface of the soft palate. This part of the work is done deliberately, and both tonsils are prepared for operation before an incision is made. I have used this method for over three years and have never seen any symptoms of poisoning such as an injection of even a small amount of cocain usually produces. Both tonsils are dissected free from the pillars before either is removed with the snare. In most cases the only part of the operation that is at all painful is when the snare is being used. For this reason it is important that the difficult part of the operation, the freeing of the tonsils from the pillars, should be done before either tonsil is enucleated. After a patient has once been hurt by the removal of one tonsil, he may be too nervous to allow the careful freeing of the second tonsil from the pillars. The separation of the tonsils from the pillars is accomplished with the least discomfort to the patient by using a straight scalpel, very sharp and with a rounded end. I find that in separating the left tonsil from the posterior pillar and the right from the anterior pillar, it is an advanatge to hold the scalpel in the left hand while the tonsil forceps are held in the right hand. The patient holds the tongue depressor. The snare to be used when removing the tonsils under local anesthesia should be one that can be closed instantly if desired. The slow closure of the snare is often too painful when local anesthesia is employed.

XLIV.

REPORT OF TWO CASES OF CEREBROSPINAL MENINGITIS PRESENTING NO CENTRAL NERVOUS PHENOMENA UNTIL SHORTLY BEFORE DEATH.

By T. Passmore Berens, M. D.,

New York.

Mr. X., age fifty-nine years, consulted me January 10, 1913. He complained of deafness in both ears and of pain in the right ear for a week, following influenza, from which he was convalescing. Examination of the ears revealed impacted cerumen, the removal of which restored his hearing to such an extent that he heard a whisper at three feet in the right ear and at eight feet in the left ear. The right membrana tympani was slightly reddened. He complained of headache, also of pain in the region of the right mastoid. There was no bulging of the membrana tympani, which was slightly injected, no edema, and no pain on deep pressure. The patient had two uncomfortable nights, then returned because of pain on January 12th. The temperature was 97.5°, the redness of the membrana tympani persisted. There was no bulging, no tenderness, but slight mucopurulent discharge from the accessory sinuses of the nose. His general appearance indicated a grave illness, and he was placed in bed in the Manhattan Eye, Ear and Throat Hospital, where myringotomy was performed under nitrous oxid anesthesia. The incision was not followed by discharge of pus. Nevertheless, he was irrigated with bichlorid 1/10,000; and in the course of six hours a profuse discharge of pus appeared in the canal. The infective organism was the staphylococcus. For the two following days the temperature was 99° and the discharge very profuse. On the third day tenderness developed over the tip and antrum of the mastoid, with slight drooping of the superior wall of the external auditory canal. Headache persisted and the patient seemed unduly apprehensive.

Under gas ether anesthesia a simple mastoid operation was performed January 18th. The mastoid process, which was extremely dense, with few cells, contained much pus. Softened bone, which was adherent to the knee of the lateral sinus, was removed. The tegmen of the antrum was softened and adherent to the dura. The wound was packed with iodoform gauze, and the patient was returned to bed in good condition.

The case progressed satisfactorily for two weeks, when there was an unexplained rise of temperature to 102°. There was no chill, no sweating. The wound was clean and healing nicely. That night he complained of marked pain in the muscles of the calf of the right leg. There was no redness, but there was tenderness. The next day pain developed in the muscles of the left calf; and there was considerable pain in the sciatic notch on this side, with backache. The tongue was deeply furred. There was marked indicanuria. The temperature was 99°. Free purgation and aspirin controlled the pains to such an extent that the patient went to the toilet and walked about the hospital. On February 4th (the seventeenth day after operation) the temperature rose to 101°, and there was an exacerbation of his pains. He complained again of headache, and that it hurt his eyes to read. Dr. Frank Van Fleet pronounced the eye grounds to be normal. The temperature then became irregular, ranging from normal to 102°. The pain left the legs and centered in the sciatic notch and in the lumbar region. There was no Kernig, no Babinsky. The patellar reflex was normal. There was no stiffness in the neck. Dr. A. B. Duel was called in consultation, and confirmed my findings, viz., a clean, healing wound, with nothing but the fever pointing to involvement of the central nervous system. Dr. Chas. H. Richardson confirmed my diagnosis of migrating muscular rheumatism. Free purgation and large doses of aspirin again gave considerable relief. The patient insisted on visiting the toilet, and seemed quite able to do so. The temperature continued to be irregular, ranging from 99° to 102°. The pulse varied with the temperature from 72 to 84; respiration 18 to 20.

On February 12th (the twenty-fifth day after operation) the patient was comfortable, and much less apprehensive than at any other time during his illness. The temperature was 100°, and he presented every evidence of a beginning convalescence.

In the evening of this day he went unaided to the toilet, which was adjoining his room; and told me that he felt better than he had felt for two weeks. At about midnight his temperature rose to 102°; pulse was 108, respirations 104, pupils irregular. He gradually sank into unconsciousness, until at 5 a. m. (on the twenty-sixth day after operation) he was comatose. I opened the original mastoid wound. It was nearly healed. The lateral sinus seemed healthy. The dura was exposed and incised. The wound through it filled immediately with a great hernia of the brain. Careful search with a brain knife failed to reveal pus in the brain. A lumbar puncture brought forth a large amount of creamy fluid under great pressure. The patient died in a few hours, without having regained consciousness.

Autopsy was not allowed, but extensive examination through the wound failed to reveal an abscess. Much creamy fluid was recovered from the ventricles.

During the course of the illness there were frequent blood counts and frequent blood cultures—all negative. Widal and Wassermann were negative.

Examination of the cerebrospinal fluid resulted in the following report:

"Appearance, mucopurulent, with considerable admixture of blood.

Copper reducing substance, absent.

Lactic acid test, strong reaction.

Cytologic Examination.—About 10 per cent bulk of pus cells on centrifuging. These cells were largely polymorphonuclear leucocytes, with a few lymphocytes and endothelial cells.

Bacteria.—In the stained smear were many chains of streptococcus mucosus capsulatus, with an unusually heavy capsule, so heavy as to leave their identity in doubt until stained by Gram's method. These grew freely on blood serum.

Stain for tubercle bacilli, negative.

(Signed) J. G. CALLISON, M. D.,
Pathologist."

G. T., male, age nineteen years, was admitted to my service in the Manhattan Eye, Ear and Throat Hospital December 5,

1912, with the following history: When an infant he fell, striking the back of the head. He was a delicate child, and did not develop well physically or mentally. The lack of mental development continued and he became an idiot. During early childhood he had measles, with a discharging ear which supposedly healed. Scarlet fever developed a few years later, accompanied by a discharging right ear. The discharge continued until the time of admission. No more complete history could be obtained because of the extreme ignorance of the patient's relatives.

Examination of the right ear revealed a large mass of spongy granulations nearly filling the canal; mastoid tender; deafness; no vertigo; no nystagmus. The temperature was normal.

A Schwartze-Stacke mastoid operation was performed under gas ether anesthesia by Dr. S. McCullagh. The external auditory canal, middle ear and attic were found to be filled with granulations, and the ossicles were absent. The position of the dura was unusually low, and the lateral sinus was far forward. The antrum was absent. There was a large exposure of the dura and of the lateral sinus. The wound was packed and the patient was returned to bed. The next day the temperature rose to 102°, the patient became restless and complained of pain in the ear and in the back of the neck. There was no suggestion of central nervous involvement. The general condition was good. The temperature dropped to normal on the third day, and for three days following pursued an irregular course, ranging from 99° to 101°. On the fifth day the temperature rose to 103°. The wound looked healthy and clean. The patient remained restless and persisted in rising from his bed. No rigidities were apparent. Babinsky and Kernig symptoms were absent. The eye grounds were normal. The blood culture was negative. Blood count: Leucocytes, 18,100; large mononuclear leucocytes, 5 per cent; small mononuclear leucocytes, 9 per cent; polynuclear neutrophiles, 86 per cent.

For the three weeks following, the temperature ranged irregularly from 100° to 102°. Other symptoms, though sought for, were not found. The eye grounds remained normal. The restlessness continued and the boy persisted in leaving the bed and in walking about. Typhoid and malaria fevers were elim-

inated by blood examinations. Many of my colleagues in the hospital saw the case in consultation. Dr. Hutton, the attending physician, made this note on the 20th of December: "Deep-seated suppuration; if intracranial, so located as to give no definite localizing signs. Heart, lungs and abdomen negative."

On December 28th the boy was found wandering in the hall of the hospital. On the following day there developed a fleeting external strabismus, and on the operated side slight facial paralysis. Ankle klonus and exaggerated knee jerk demonstrable, and Kernig's sign positive. Neck slightly resistant. The eye grounds were normal. A lumber puncture revealed the cerebrospinal fluid, which was under pressure, to be almost creamy in color and consistency.

The evidences of central nervous involvement did not increase. A few hours before he died he arose from his bed and appropriated articles belonging to other patients. He sank into coma an hour or two before death.

Autopsy revealed an unusually well developed cerebrospinal leptomeningitis.

Examination of the cerebrospinal fluid resulted in the following report:

"About 15 cc. recovered. 10 cc. sent to laboratory.
Appearance, greenish, creamy, mucopurulent.
Amount of sediment, 2½ cc. in 10 cc.
Copper reducing substance, absent.
Lactic acid test, very marked reaction.
Cytologic examination—Cellular elements are mostly polymorphonuclear leucocytes, with some lymphocytes and endothelial cells.
Bacteria.—Large numbers of either streptococci or pneumococci, with some bacilli resembling B. pyocyaneus. These cocci were culturally proved to be streptococcus mucosus.
Stain for tubercle bacilli negative.

(Signed) J. G. CALLISON, M. D.,
Pathologist."

The postmortem examination, conducted by Dr. Kopetzky, revealed the spinal cord covered with a thick, creamy exudate, which extended into the ventricles of the brain. There was no involvement of the lateral sinus. The dura was normal over

the seat of the mastoid operation. The internal auditory meatus was filled with granulation tissue and pus.

I have reported these two cases not because cerebrospinal leptomeningitis of otitic origin is a rare disease, but because in my experience, covering twenty-five years of otologic practice, I have not seen a single other case which did not present long before death some symptom other than fever pointing to involvement of the central nervous system. I confess myself at a complete loss to explain the lack of symptoms here presented. Brieger, quoted by Preysing, in the Transactions of the German Otological Association, 1912, says:

"In many cases of suppurative meningitis we see all the symptoms disappear after opening the labyrinth and after repeated lumbar puncture. I have seen such cases, which appeared almost in the last stages, seemingly completely recover. They got up out of bed, walked about, felt perfectly well; and we were astounded to see that within a few hours after a conditions of apparent complete well-being death should supervene. Autopsy revealed that in some cases there occurred in parts of the subarachnoid space, inaccessible to lumbar puncture or to other exploratory methods, a widespread plastic infiltration, while in other cases there existed, in addition to these localized infiltrations, diffuse inflammation of the meninges."

XLV.

CHOICE OF TREATMENT IN CHRONIC SUPPURATIVE OTITIS MEDIA.*

By George L. Richards, M. D.,

Fall River.

What is intended to be discussed is not the details of any operative or other treatment, but rather to what conclusion we should come with reference to the choice of treatment in cases of chronic purulent otitis.

First, in children. The records of all the ear clinics where children are treated show that many of them seek treatment for chronic discharging ears. On a history being sought, none will be obtained at first, but careful, skillful questioning will bring out the fact that the ear discharge has been the result of a cold, or has followed one of the exanthemata, measles or scarlet fever. If stated to be due to frequent colds, the first thing to ascertain is the presence or absence of adenoids or enlarged tonsils, and then to correct any abnormal pharynx or nasopharynx condition which may be evident or causative. Quite a number of cases clear up promptly with cessation of the discharge. Those which do not, and especially those due to the exanthemata, usually show on examination a considerable loss of the drum membrane and adhesion of the ossicles to the inner tympanic wall. Such cases should be treated for a while conservatively and carefully by such thorough cleansing methods as appeal to the individual treating the case. Unfortunately, this cleansing can only be done by the physician himself or by a nurse, and almost never by the patient or friend. This necessitates frequent visits to the physician's office or clinic, and is one of the drawbacks to the conservative treatment of chronic ears. Nevertheless, a large number of these cases will clear up, the discharge stop, and

*Presented before the American Laryngological, Rhinological and Otological Society, at its meeting held in Washington, May, 1913.

the hearing improve. Nature left to herself, if she has any reasonable opportunity for drainage, will usually wall off the diseased area from the sound area, and in the case of bone, either casts off the disease or forms a sclerosed wall between the diseased tissue and the sound area. While it is no doubt true, as William McEwen wrote many years ago, "that all cases of suppurative ear trouble are potential volcanoes," the fact remains that only relatively few of the actual number of cases either come to radical operation or die as a result of the disease.

What next is to become of these cases of suppurative ear trouble which, after long and careful cleansing treatment, do not subside and do not clear up? What is to be recommended? Some type of operation, and, if so, what? In the choice of this, I think due consideration should be given to the question of how much hearing the child has—and many times the hearing is very good—and also to his or her ability to continue treatment. If the hearing is nearly or completely gone, the socalled radical operation is the operation of preference, as it comes nearest to complete surgical removal of the diseased material. Even here a more or less long course of after-treatment may be necessary, and it cannot be stated with absolute certainty that such an operation will always be successful in completely stopping the discharge. Many a case subjected to radical operation has occasional times when it suppurates, but as the drainage is good there is little danger from this, and if cases operated radically are seen occasionally there is little trouble.

When there is a fair degree of hearing and the major portion of the drums remains, I think some type of conservative surgical operation should be adopted, providing cholesteatoma is not present. By a conservative type I mean those advocated by Heath, Bondy, and Stacke, in which the drum membrane and ossicles are allowed to remain, and the tympanic cavity is drained from behind through the external canal. It seems to me that in America we have not yet sufficiently tried out this type of operation. I am aware that the socalled Heath operation has not fulfilled its expectations in the hands of others than its originator, but that is no reason for not continuing the search for a satisfactory conservative operation. While it is no doubt true that in some cases after a complete

radical operation the hearing is not materially damaged, or may even be improved, yet reasoning from an anatomic standpoint it seems impossible that the hearing should be otherwise than usually made worse, and this I think will always be found to be the case if the affected ear is tested sufficiently carefully both before and after the operation.

How long ought a child's ear to be treated conservatively before some type of radical operation is performed? This is one of the hard questions before us. In our clinics patients with chronic ear discharge are asked day after day to return for what to the patient or parents or others seems a continual wiping out, without much result, and in the case of patients who come to our office and pay our fees, it seems to many of them a long drawn-out, expensive procedure, with, in many cases, nothing or little in the way of result. And when finally we suggest to them that after all an operation is required, they not infrequently and very naturally ask, "Well, if that is the case, why was it not done to begin with?" So that before beginning any conservative method of treatment an explanation, sufficiently simple to be understood by the patient, should be given him so as to forefend oneself.

The question will very naturally be asked, "If the ear becomes dry, will it stay dry and will I hear?" This, it seems to me, is largely a question of whether moisture, that is, water, can be kept out of the ear. After an experience of some twenty years in treating these cases, I am firmly of the conclusion that the drier we can keep them, the better and more rapid the results. You all know how difficult it is to satisfactorily dry an ear when you have the patient before you under most favorable conditions. When syringing the ear is ordered for home use, the ear is never dry, and it is practically impossible for anyone to properly dry the ear except under good illumination. What then is the result? A moist cavity at temperature of the body, and the microorganisms already present are only favored in their development by the conditions provided.

Second, in adults. When we turn to adults, we are confronted with two classes of chronic suppurative middle ear: one with, and one without cholesteatoma. Those without cholesteatoma may be handled somewhat the same as I suggested in children. An endeavor, first of all, is to be made to obtain a cure by conservative means, which with many

things at our command for local use, and the careful cleansing and mild curetting which it is possible and permissible to do under good illumination, will, in most cases, be followed by good results. Here again the caution with reference to water and bathing is necessary. The question of hearing is not usually of great importance, unless both ears are involved, since if the adult has one good ear, and seeks treatment for discharge for the other, which is practically deaf, he does not usually expect the restoration of hearing in the affected ear, which is apt to be poor, and from which the discharge has usually continued for months or years before relief is sought. A radical operation is then to be advised in many cases.

The question of conservative treatment in the cholesteatoma case depends entirely on whether the opening is sufficiently large to allow the discharge of the cholesteatomatous mass. Where this is the case, and where the ear can be kept dry, even a cholesteatoma not infrequently can be successfully treated by conservative means. Where the opening is small and the cholesteatoma is moist, nothing short of a radical operation is to be advised. In the noncholesteatoma cases, most of them can be treated conservatively with results which are fairly good. In adults, as in the case of children, if conservatism is not followed after a reasonable time with good results, then the question of a radical operation is to be considered. It is surprising, however, what a large number of cases can be cured by careful attention to conservative methods. While I have no exact statistics, I am of the opinion that most of the cases of severe complications have occurred in patients where the suppurative otitic trouble has not been treated, but where it has been allowed to go on without much attention on the part of the individual until infection, through some channel or other, has reached either the brain, mastoid or lateral sinuses.

I have been interested to look over the statistics reported from some of the large ear hospitals with reference to the proportionate number of cases of purulent ear trouble which seek relief, as compared with the number of cases of operations done in these hospitals. The New York Eye and Ear Infirmary reports for the year 1911, 1283 cases of chronic suppurative otitis media, with 110 cases operated upon, a proportion of 8.5 per cent. The Manhattan Eye and Ear In-

firmary reports of 1906-07-08-09-10 gives about the same results, 7350 cases, of which 706 were operated upon, or 10.4 per cent. The Massachusetts Eye and Ear Infirmary reports, so far as radical operation are concerned, for the years 1909-10-11, 6507 cases of chronic suppuration treated in the out-patient department, and with 80 radical operations, or 1.25 per cent. So that at least not over one in ten of those who seek relief, even at our large hospitals, are operated upon, and it seems to me that if thorough conservatism is applied, that in one's private practice more than nine out of ten can be cured without operation. Certainly the pendulum at the present moment seems to have swung back from the time when operation was suggested for nearly every case of chronic suppurative otitis to a somewhat middle ground, where cases manifestly demanding it are operated upon, while many others are cured, or at least the process brought to a standstill, without operation. In the final analysis each case must be judged on its own merits, due consideration being given to all the factors in evidence.

XLVI.

RADIOGRAPHIC FINDINGS ILLUSTRATING THE ANATOMIC DEVELOPMENT OF THE MASTOID BONE.*

(STEREOPTICON DEMONSTRATION.)

By WILLIAM H. STEWART, M. D.

NEW YORK.

On December 29, 1910, I demonstrated before the Section on Laryngology and Rhinology a series of radiographs showing the development of the frontal sinuses. The cardinal points of the paper were the very early evidence of the frontal sinuses and the wide variations in size, location and existence. At the suggestion of your very able chairman, I have attempted to take up this line of work with the mastoids. I was stimulated by the general belief that the mastoid cells, under ten years of age, were represented by a spongy cancellous layer of bone, lying between the inner and outer table of the skull, called diploe.

Dr. Sidney Lange, one of the earliest workers on radiographic examination of the mastoid, says in his original article, read before the American Roentgen Ray Society on September 23, 1909: "Upon the mastoid skiagrams of children under ten years of age the mastoid appears entirely spongy and diploetic, there are no visible cells and the tip is undeveloped. The tip at this time consists simply of an inner and outer table with little cancellous bone between and shows no structure upon the skiagram. From ten to fifteen years of age the mastoid takes on the pneumatic characteristics, larger cells appear and grow downward toward the tip, its diploetic structure finally giving way to the more or less pneumatic adult type."

*Presented before the New York Academy of Medicine, Section on Otology, May 12, 1913.

Gray says: "The mastoid cells, like the other sinuses of the cranium, are not developed until after puberty."

Piersol says: "The mastoid process becomes fairly distinct in the course of the second year; it develops greatly about the time of puberty, when it becomes pneumatic. This may occur much earlier. J. J. Clark has seen it wholly pneumatic several times before the tenth year; once at three and one-half years of age."

The results of my investigations have been partially successful in demonstrating:

1. That we frequently have distinct and well formed pneumatic cells as early as two years.

2. That there is a wide variance in the character, location and extent of the mastoid cells under ten years.

3. That given six children of any age, say five years, no two will have mastoids alike.

4. That the generally accepted view that the cells are of the same character on both sides is not true in all cases—my experience with the normal will show that while the majority have a great similarity on both sides, that in about 15 per cent there is considerable variance in the character.

5. That the mastoid cells of the pneumatic type may extend into the occipital bone back of the sigmoid portion of the lateral sinus, into the squamous portion of the temporal, and above the external auditory canal to the root of the zygoma.

7. That with a fairly intelligent child the radiographic examination will clearly show the character and location of the mastoid cells in their relation to the lateral sinus, the external auditory canal and the tegmen tympani.

It may be interesting to quote the following observations in adults:

In Zuckerkandl's investigation of 250 temporal bones, the mastoid was found entirely pneumatic in 36.8 per cent. and wholly diploetic in 20 per cent. The remaining 43.2 per cent were mixed, the diploe being at the point of the mastoid and the cells above; neither size nor shape indicates its internal structure. This is somewhat in variance with Piersol, who quotes Randall in the study of 1000 temporal bones, in which he found scarcely 2 per cent of mastoid cells could be classed as diploetic, and only some 10 per cent as combining a notable amount of diploe with pneumatic spaces.

TECHNIC.

These radiographs were all taken of normal children from the age of one month to fifteen years, and I beg your indulgence if they seem comparatively poor, as it is no easy task to control a healthy child sufficiently to properly examine the mastoids with the X-ray. Extreme patience and indulgence are necessary. Success can only be achieved when you have the confidence of the child. This may be acquired in many ways. The method I pursued was as follows:

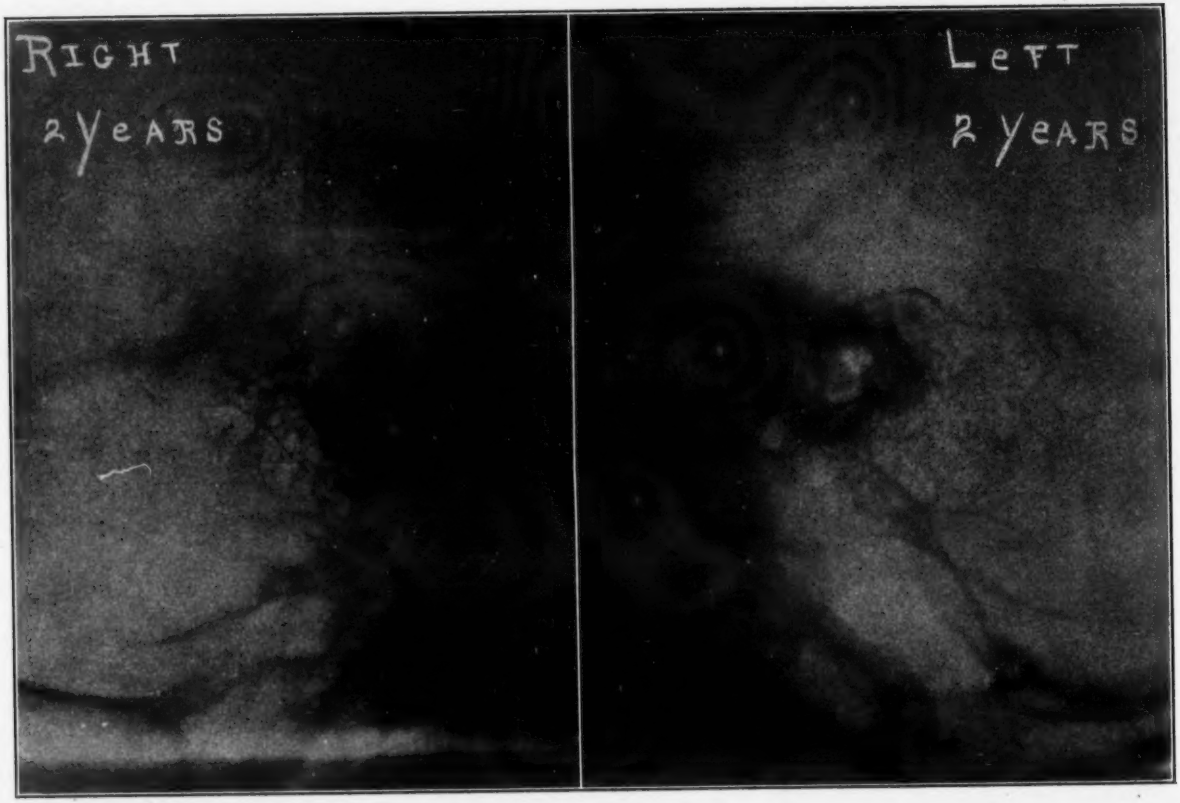
After repeated failure in not being able to obtain absolute immobility, I conceived the idea of having afternoon tea parties; one of the pleasures of this gathering was to have their "picture took." I have strenuously fought being relegated to a photographer; in this case I was obliged to be just an ordinary picture man. The children were gathered around so that they could all see the camera "shoot," that it did not hurt and that you must hold your head still.

The child was laid on the table on its side, the head resting on the plate, placed on a wooden inclined plane of 25 degrees, the top of the plane at the neck; this brought the plate in direct contact with the mastoid region, the ear being pushed forward; a line drawn from the glabella through the middle of the tip of the nose to the symphysis of the chin was parallel to the upper surface of the table, which was supposed to be level. An X-ray tube of medium penetration with a vacuum that would take about 55 milliamperes was then adjusted in the tube stand, having a cone with an outlet of about four inches diameter attached; the axis of the cone being continuous with the central ray of the tube.

The cone, set so that the ray would be directed from above 10 degrees and behind about 15 degrees, was then moved over the head so that the axis would be centered at the point just below the parietal eminence on the upper side. An exposure is then made of about two seconds. This will, in the majority of cases, give us good radiographs of the mastoid with a minimum amount of distortion; it may be necessary to vary the angle somewhat, as we meet with different shaped heads.

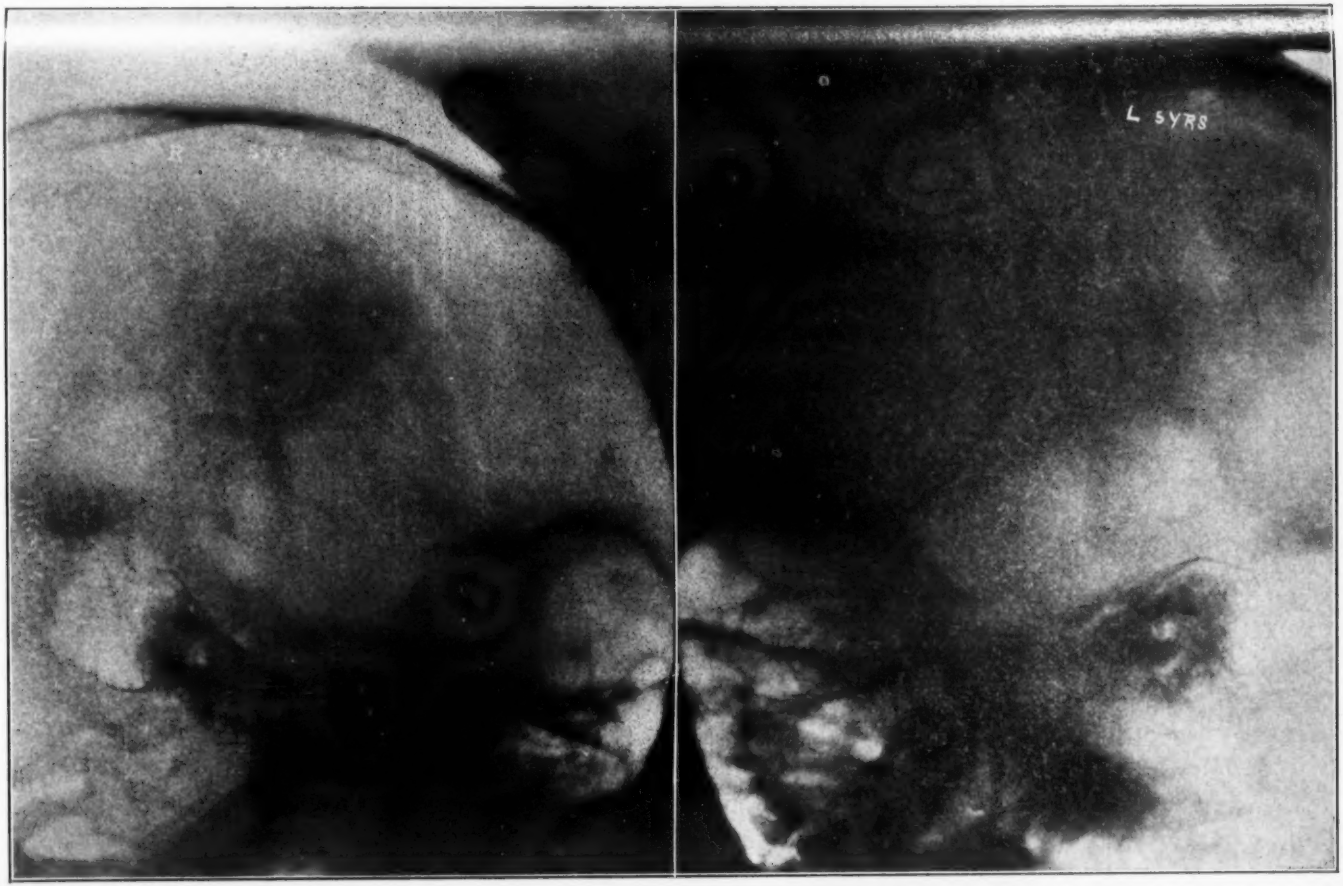
One of the difficulties we have to contend with is to procure skiagrams of uniform density of both mastoids. My observations are much the same as Lange's. He says: "As a rule, a slightly underexposed plate is best to study."

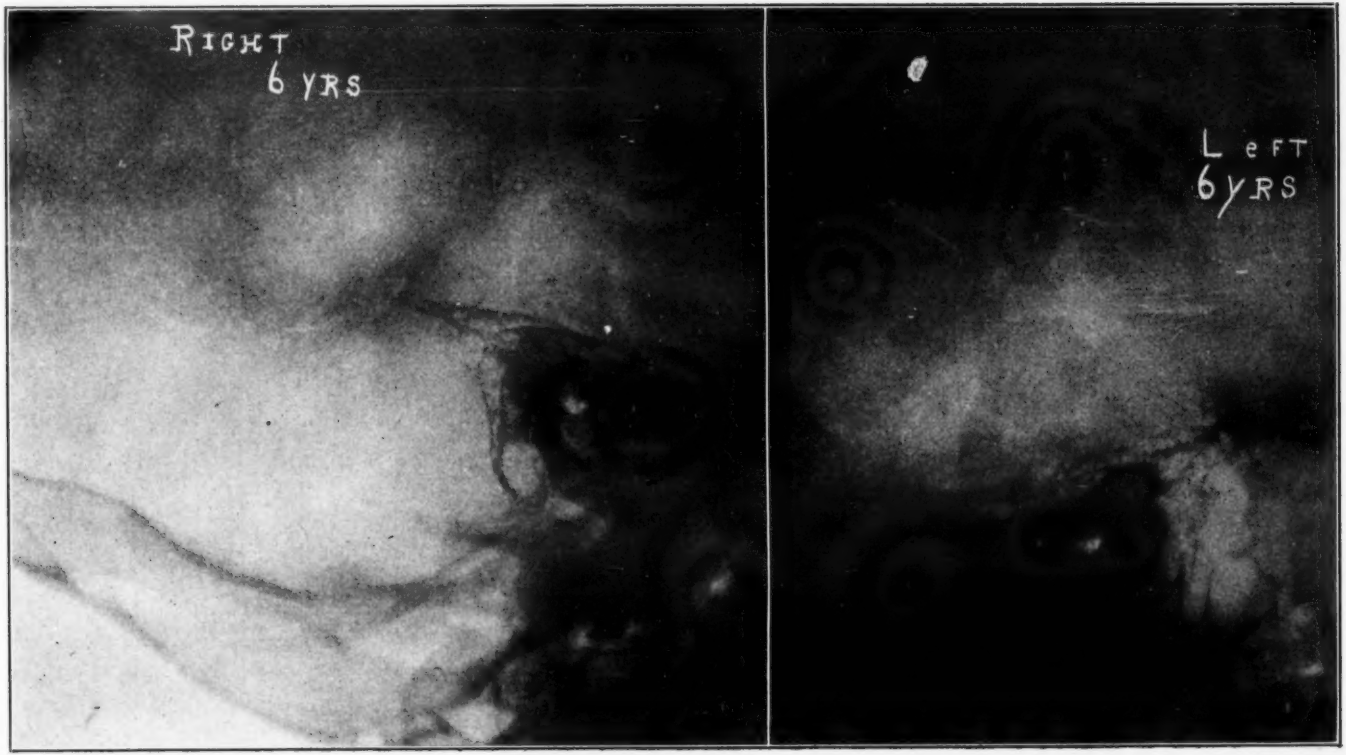
While examining numerous little patients with supposed fracture of the skull, in which a portion of the routine was radiographs of the occipital region, I frequently observed that the mastoid cells were well brought out in their transverse diameter and that the mastoid antrum was frequently seen. I have now adopted this as a part of my technic in mastoid cases—examining my patients first in this anteroposterior and then both sides in the posterolateral oblique positions.

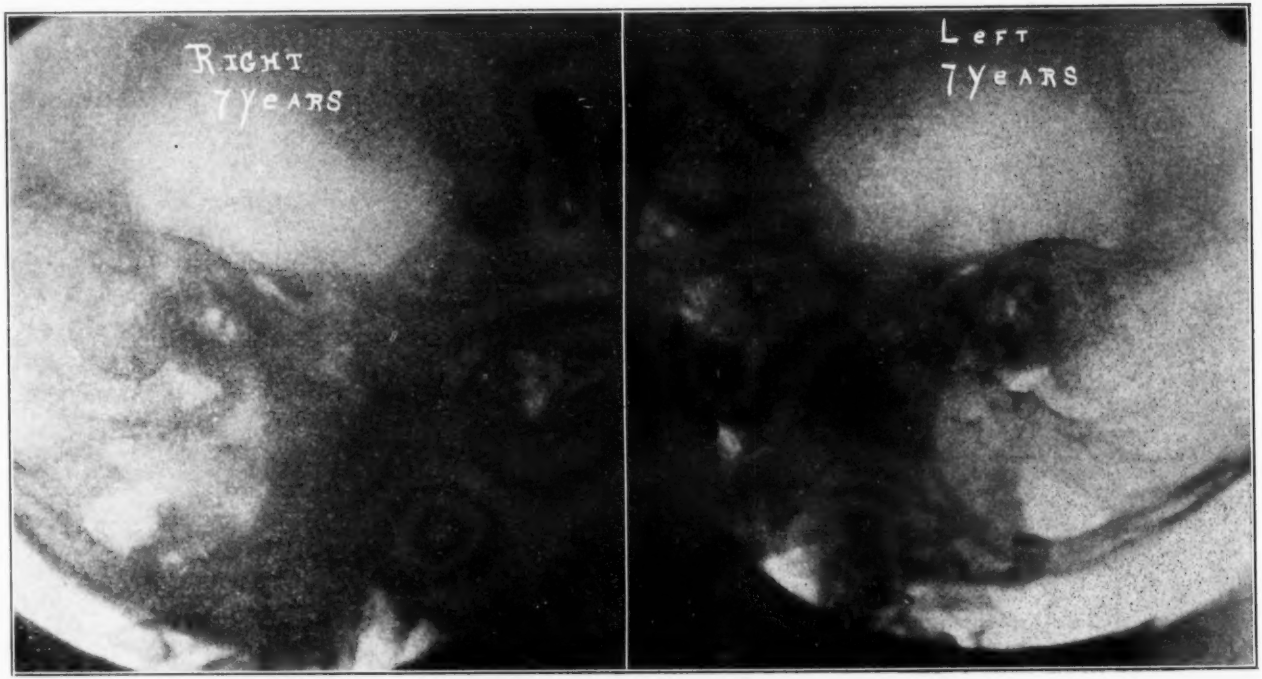

RIGHT
2 YEARS
LEFT
2 YEARS

RIGHT
4 YEARS
LEFT
4 YEARS

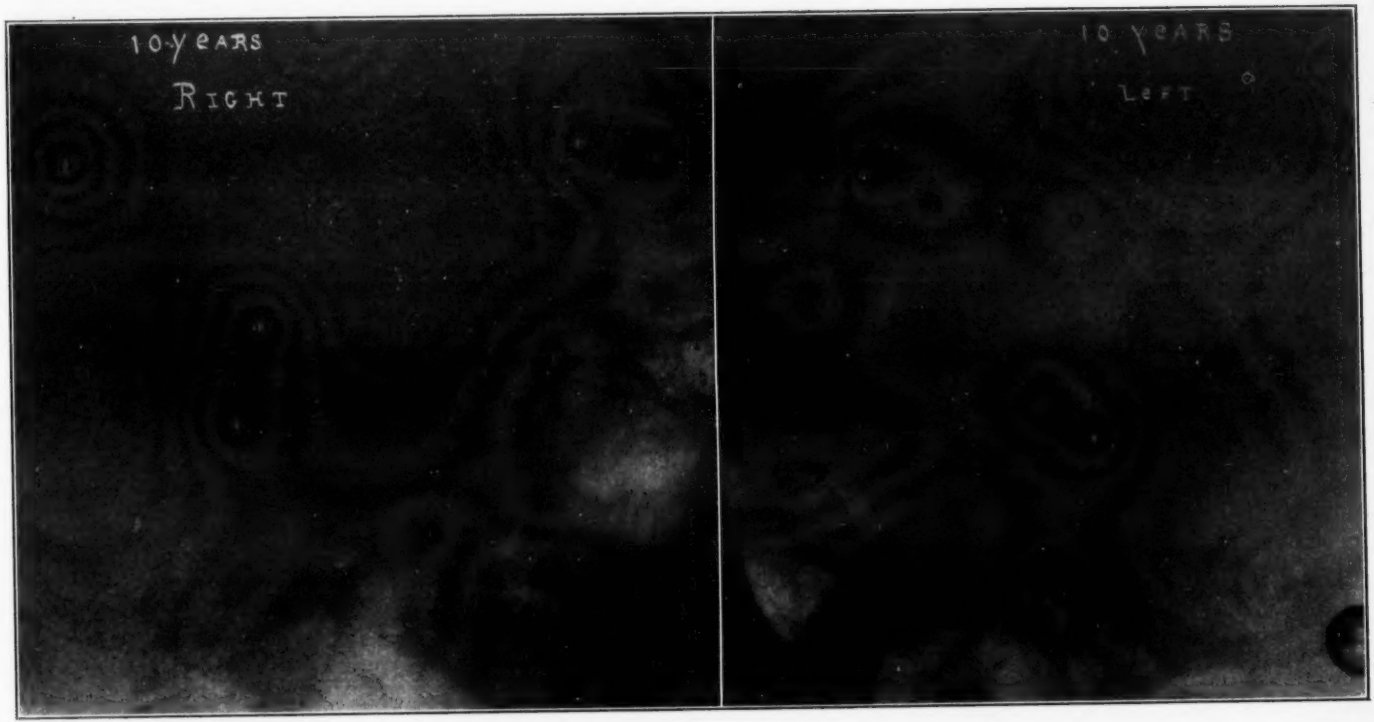
LEFT
4 YRS
RIGHT

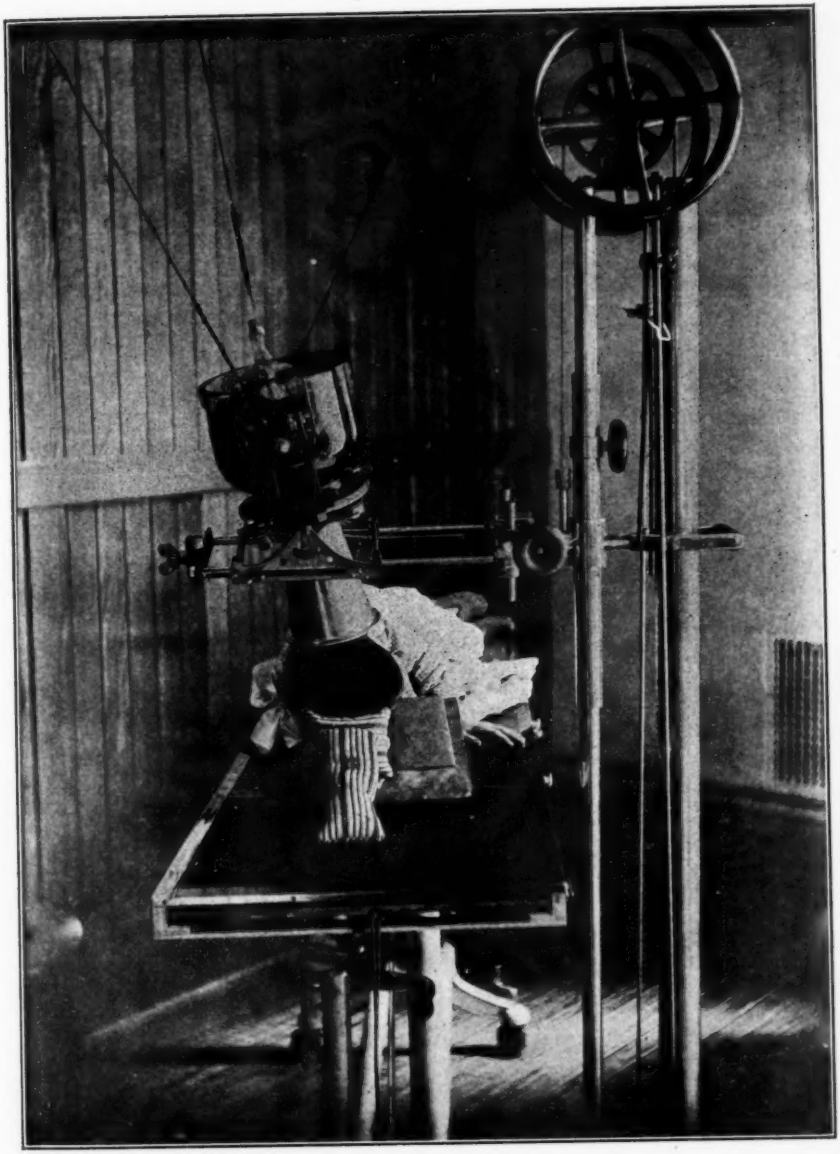

L 5YRS


RIGHT
6 YRS
LEFT
6 YRS


RIGHT
7 YEARS
LEFT
7 YEARS

RIGHT
8 YEARS
LEFT
8 YEARS


10 years
Right
10 years
Left



XLVII.

TOPOGRAPHY OF THE TYMPANIC CAVITY.*

By John A. Cavanaugh, M. D.,

Chicago.

The subject which is chosen for this thesis is one which most of our modern textbooks touch upon in a general way, and its importance is confirmed by personal observations on wet and dried specimens. Some of these specimens are photographed and are reproduced in this paper.

The development of the middle ear should be understood in order to know how the surrounding structures are sometimes invaded by diseases of this cavity; therefore, a brief résumé may not be amiss. The temporal bone is developed by ten centers, exclusive of those for the internal ear and the ossicles: one for the annulus, one for the squama zygomatici, two for the styloid process, four for the petrous, and two for the mastoid. Huxley declares the centers are more numerous and gives a somewhat different classification. The mastoid and petrous portions are united before birth; however, an imaginary line drawn in a vertical plane close behind the stylomastoid foramen and at right angles to the whole petromastoid portion, marks the union between these two portions. We have at birth three ununited portions of the temporal bone—the squama zygomatici, annulus and petromastoid parts—which are held together by bands of connective tissue. Ossification usually takes place between the two upper extremities of the annulus and adjacent portion of the external table of the squama, so that after a certain period the temporal bone separates into two portions—the united annulus and squamous portion and petromastoid portion. During the first year the bony union of these three parts, namely, the annulus, squama and petromastoid, are very imperfect. The annulus at birth (Plate I, Fig. 1) is like a plain ring, only that the upper pos-

*Read before the Chicago Laryngological and Otological Society, February 18, 1913.

terior part is wanting, leaving a gap which is filled in by the outer table of the squama.

As a child develops there is an outward growth of the outer edge to form the future external auditory meatus (Plate I, Fig. 2-C and 2-A) and an inward growth from the inner edge to help form the floor of the middle ear. The horizontal portion of the squama divides into an outer and inner table, the outer forming the external wall of the attic, while the inner helps to form a part of the roof of the middle ear (Plate I, Fig. 3-A and B). The petrous portion forms the inner and posterior walls of the middle ear.

The tympanic cavity is the largest cavity of the temporal bone in the larger percentage of cases, but anomalies may exist where an unusually large antrum or mastoid cell is present (Plate I, Fig. 6-A; Plate II, Fig. 10). I have in my collection a temporal bone wherein there is a cavity in the petrous portion extending from above the inner part of the internal auditory meatus, just internal to the superior and posterior surface, to the Glaserian fossa: it measures 20 mm. long, 9 mm. wide, and from 5 to 10 mm. deep (Plate I, Fig. 6-A). The tympanic cavity is of cuboid shape, the walls being very unequal. It is formed by the pars petrosa, pars squamosa and pars tympanica, though the pars petrosa forms the greater part. This cavity contains air, reduplications of mucous membrane, muscles, ossicles and the ligaments of the ossicles. This cavity we describe usually as having a floor, roof, an anterior, posterior, external and inner walls. If we were to consider the median plane of the skull, these are not quite accurate, for the inner wall lies at an angle of about 45 degrees from the sagittal plane.

The roof is concave downward from without in, and generally increases in height as it passes from the processus cochleariformis backward to become the roof of the antrum (Plate I, Fig. 5-A and B). It is formed mainly by a little lamina of bone which projects outward and forward from the superior surface of the petrous bone to join the inner table of the squama as it turns inward; this marks a fissure, the petrosquamous, which in children lies over the tympanum, but in adults is usually found a little external to this roof. It has no special anatomic features of interest, but it is of surgical and pathologic importance, since infection may pass through this

area from the middle ear invading the meninges. Occasionally a dehiscence (Plate I, Fig. 4-A) exists, and the dura is in direct contact with the lining of the middle ear space. This fissure usually closes at the fifth month of life (Kopetzky). Examining 765 skulls, such defects were noted one hundred and sixty-seven times (Burkner). Körner found these dehiscences present eighteen times in 209 skulls, and also found in some cases the tegmen to be several millimeters thick, and where this exists, small air cells are adjacent to it. According to R. Johnson Held, the tegmen usually contains numerous foramina for the passage of small blood vessels, and at times has a cribriform appearance; he has found dehiscences twenty-nine times.

The floor is formed by a projection outward of the inferior surface of the petrous bone, which Gruber calls the fundus tympani, as it forms the greater part of the floor of the tympanum. This floor is also formed by the inward growth of the inner edge of the annulus. It narrows as the descending outer and inner walls approach each other. It lies a little below the floor of the external auditory meatus. It is pushed upward, forward and outward in some cases, while in others upward, forward and inward, at its posterior part, due to the encroachment of the jugular bulb (Plate I, Fig. 7-C, and Plate II, Fig. 8-F). There is sometimes a dehiscence in this area which allows the jugular bulb to be seen from the tympanum, and even gives a bluish tint to the lower part of the tympanic membrane in the living subject as seen through the speculum. Sohier Bryant, in the *Archives*, 1905, says: "In children the jugular fossa is below and not behind the tympanum." I have found both positions present. The bulb on the right side is usually larger, consequently there is more of an elevation of the floor, and, in my opinion, more reason for the presence of a dehiscence. I find, however, no literature verifying this idea.

The possibility of the existence of a dehiscence should always be remembered when performing a paracentesis of the drum membrane. In such conditions the bulb is always liable to become infected in suppurative conditions of the middle ear. Carl Koller, in *Archives*, 1904, reports such a case. Turgens (Warsaw) reports a hemorrhage from the bulb after the use of strong caustic in the middle ear. Lafarelle reports a case

of a diverticulum of the tympanum from the floor, which extended downward, and was of the size of the tympanum, the walls of which were very irregular.

There is a small aperture in the floor, close to the inner wall, for the passage of Jacobson's nerve (Plate II, Fig. 8-C). In the macerated specimens this surface is usually very uneven, due to irregular cellular spaces.

The anterior wall can hardly be said to exist, for its place is taken up mostly by the tubal entrance and the tensor tympani muscle, the latter entering the tympanum above the tube (Plate I, Fig. 6-B and C). A small portion of the anterior wall does exist beneath the lower lip of the eustachian opening and floor of the tympanic cavity, where the fundus tympani turns up and separates the carotid artery from the middle ear. Sometimes this bony wall is wanting, thus allowing the carotid artery to be in contact with the lining of the middle ear. A number of cases are reported of fatal hemorrhages due to trauma of the anterior wall. Where a dehiscence is caused by an erosion, it is usually found at the vertical and horizontal part of the canal. Politzer attributes this to inflammatory softening due to pus which surrounds the artery, and which causes a diminished resistance; the repeated concussions of the blood wave finally results in a perforation of the anterior wall.

Another important anatomic feature of this anterior wall is around the lower lip of the tubal entrance, and consists of small cellular spaces known as Koerner cells (Plate II, Fig. 9-D), which become infected, and in some cases produce constant obstinate otorrhea. The failure to eradicate these cells thoroughly at the time of a radical operation is often the cause of constant trouble. Urbantschitsch, in the third edition of his textbook, speaks of finding in certain temporal bones cellular spaces in the vicinity of the tympanic orifice of the eustachian tube, which led to a system of pneumatic cells, and which in turn spread to the tip of the petrosal pyramid, and through dehiscences communicated with the middle fossa of the skull. I have one such specimen in my collection (Plate II, Fig. 10).

The posterior wall is absent in its upper part, due to a variable sized opening of a somewhat triangular shape, with its apex pointing downward (Plate II, Fig. 11-A). Its base is about 5 mm. wide, and from its base to the apex measures

about 6 mm. The apex of this triangular opening lies just internal and above the upper end of the posterior arm of the annulus (Plate II, Fig. 12-E), and is called by some authors (Davis and MacEwen) the iter or aditus which leads into the mastoid antrum. Most of the literature I have consulted refer to a small recess just below the floor of this socalled accessory opening of the tympanum, the sella incudis, for the reception of the short process of the incus. This statement, according to my observation, is incorrect, as the short process rests on the floor of the iter or aditus close to the posterior wall, but not on the posterior wall. Close to the inner wall and below this opening is a small projection—the pyramid—with a tiny opening at its apex, from which issues the tendon of the stapedius muscle which arises from a cavity in its interior (Plate II, Fig. 13-D). From the inner and lower side of the pyramid I have found in most specimens a delicate thread-like bridge of bone extending to the upper edge of the beginning of the promontory. Below, at a point where the floor and posterior walls seem to meet, is a dense hard projection, which is the tympanic end of the styloid process. I have found in some cases, around this projection, numerous cellular spaces, which are important to remember in radical mastoid surgery (Plate III, Fig. 14-A). Through this wall passes the facial nerve, which enters just above and at the posterior end of the oval window, and runs in a somewhat diagonal direction, downward and outward, leaving this wall of the tympanic cavity just behind the middle of the posterior arm of the annulus (Plate I, Fig. 1-A; Plate II, Fig. 8-E, and Fig. 11-B). B. Alex. Randall, in the *Archives* of 1913, reports his findings in one hundred cases, where the facial nerve lay less than two nor more than four millimeters from the back wall of the meatus, and crossed the oblique plane of the drum three millimeters back of the middle of the posterior margin of the annulus. Hugh E. Jones, in the *Journal of Laryngology, Rhinology and Otology*, of June, 1903, and H. A. Alderton, in the *Archives* of 1904, give very important data regarding the location of this nerve. To avoid injuring this structure, one should not go below the floor of the antrum where it joins the posterior arm of the annulus (Plate II, Fig. 12-E).

The external wall is formed by bone and membrane, the upper part by the external table of the squama, which contains

cellular spaces in the adult and lies over the superior meatal wall; the lower part by the drum membrane and annulus tympanicus (Plate I, Figs. 1 and 3). The lower edge of the external table of the squama for about four millimeters is not joined by the annulus (Plate I, Fig. 1-C, and Plate II, Fig. 12, between C and E). This area, which is in the upper and posterior part of the drum, and lies just anterior and slightly below the opening into the mastoid antrum, is where the periosteal covering of the upper part of the meatus passes into the inner surface of the external table of the squama, thus forming the periosteal covering of the outer wall of the antrum and attic, and allowing subperiosteal infections to travel into the external meatus from the attic and antrum at this point. Swelling here indicates attic and antrum inflammation. The annulus in most subjects is horse-shoe in shape, but cases are reported where the osseous circle is complete. Hyrtl has reported a case wherein two tympanic rings existed. The annulus forms only a small part of the external wall; on its concave side there is a sulcus—the sulcus tympanicus—for the insertion of the membrana propria (Plate I, Fig. 2-D). At the upper and posterior part, where the annulus is missing, the sulcus is also missing, and this area is known as the notch of Rivini (Plate I, Fig. 1-C, and Plate II, Fig. 12, between C and E). The annulus is placed obliquely, inclining toward the anterior wall and floor of the meatus, forming with the superior and posterior walls an angle of about 145 degrees, while with the floor and anterior wall it forms an angle of about 35 degrees. The annulus has a somewhat spiral shape, and its anterior extremity makes a slight twist on its long axis, so that the sulcus of the annulus is seen from the meatus in dried specimens when held in its normal position. At the upper end of this anterior arm is a small tubercle, the crista tympanica, and above this a little groove, the sulcus malleolaris, which lodges the processus longus of the malleus. The development of the annulus along the anterior part of the lower margin of the squama forms the Glaserian fissure, which is of special interest to the otologist, as it sometimes serves as a passage by which infection may spread from the tympanic cavity into the region of the parotid gland, or may even affect the inferior maxillary joint. This fissure is formed by the crista tympanica of the annulus and the tegmen tympani which

advances to meet it; these structures draw nearer to the lower anterior margin of the squama and practically close this area; however, in the young, before this closure has taken place, and in those cases where development does not progress normally, it leaves a relatively large opening. Infection of the middle ear may travel through this opening and become the primary cause of some supposed parotid gland or pharyngeal abscess, and should be remembered.

The drum membrane is an irregular oval membrane, measuring about nine by ten millimeters (Plate III, Fig. 17). It is placed in the same plane as the annulus. The drum is concave outward at the umbo or tip of the malleus, and between that and the annulus it is convex outward, excepting in its posterior quadrant, where it is somewhat flattened. The membrane is transparent and about one-tenth millimeter in thickness. It separates the external auditory meatus from the middle ear. It consists of three separate layers: an outer cuticular, a central or membrana propria, and an inner or mucous layer. Zalewski, through experiments, found that pressure of from one to two atmospheres was necessary to cause rupture of the normal drum, which in atrophic conditions or cicatrices of the drum, infectious processes, or advanced age, lessened pressure was required. In fibrous or calcareous thickening, increased pressure was necessary. Gruber contends that the drum will stand a gradual increasing pressure of from sixty to seventy pounds.

There seems to be a diversity of opinion as to the position of the drum membrane. Most authors are inclined to believe the drum membrane lies more horizontal in the child than in the adult, while J. Pollak and Symington have agreed that the drum lies in the same plane in the adult as in the child. From my observations, I must agree with the latter (Plate III, Fig. 15 Adult and Fig. 16 Infant).

I believe that because of the high position of the auricle, the cartilaginous floor of the meatus lies almost parallel with the outer surface of the drum in children; while in the adult, because of the development of the osseous meatus, we look at the drum in a more horizontal direction. It would seem to me, if the annulus changed its position it would have some effect upon the size of the tympanic cavity, but this cavity is about the

same in size in the young and old. I have removed the bony external auditory meatus in the adult to the drum membrane, and from all appearances it seems to be in the same plane as in a child. I have also made an artificial bony external meatus by casts and placed this against the infant annulus as it would appear later in the adult, and the drum seemed to be on the same plane as the adult membrane.

The membrana tympani is composed of a tense and a flaccid part. The former is tense because of the membrana propria, which is made up of the circular and radiating fibers, while the latter, or Shrapnell's membrane, is flaccid because it is devoid of these fibers (Plate III, Fig. 17-B). The long process of the malleus, of which we shall speak later, is readily seen through the drum membrane, and at the upper part of this handle is a round projection, the short process of the malleus. From the short process of the malleus, passing upward in opposite directions to the upper end of the annulus, is the edge of the membrana tensa, which is thickened and known as Prussak's fibers, called by some authors the anterior and posterior folds, while Bezold and Siebenman call them the anterior and posterior liminal strands (Plate III, Fig. 17-A and C). The area between these two bands is known as Shrapnell's membrane. It is of the greatest importance to remember the position of the drum membrane when removing foreign bodies from the meatus or performing a paracentesis. Perforations in the various parts of the drum are also of the utmost importance, and may be divided into the dangerous and nondangerous variety. When the membrane seems to be entirely destroyed in a marginal perforation at its junction with the annulus, or where there is a perforation in Shrapnell's membrane, or in the posterior superior quadrant, it is to be viewed with suspicion as of a possible dangerous type (Plate III, Fig. 19-A); while a perforation which exists where a membrane, be it ever so small in amount, separates it from the annulus, may be classified as belonging to the nondangerous variety (Plate III, Fig. 18-A). I am thoroughly convinced that in diseased conditions of the antrum, perforations at the posterior superior part of the drum and in Shrapnell's membrane are due to secretions drawing from the antrum directly upon the drum at this point, thus causing destruction in this area (Plate III, Fig. 20-A). The

short arm of the incus forms with the outer table of the squama a trough, so to speak, which retains secretions and aids infections of Prussak's space (Plate IV, Fig. 23-A).

The inner wall looks forward and outward and is the most important of the various walls of the middle ear. It forms also the outer wall of the internal ear. From the tegmen tympani downward we observe:

First—A smooth bony projection in the upper posterior part, called the horizontal semicircular canal, which lies in a horizontal plane and passes back through the iter into the antrum (Plate I, Fig. 2-C).

Second—Below this elevation and lying in the same plane, but extending a little more anteriorly and not so far posteriorly, is another bony elevation, which marks the position of the canalis fallopii, through which the facial nerve passes (Plate II, Fig. 12-B). There is sometimes a dehiscence in this ridge, and mucous membrane only separates it from the tympanum. This is a very important surgical landmark, and its position should be thoroughly familiar to surgeons doing middle ear and mastoid operations. In infection of the middle ear where a dehiscence exists we are liable to meet with facial nerve complications, due to an exudate into the surrounding structures causing pressure. Infection may travel along the sheath of the nerve to the cranial cavity.

Third—Directly anterior and a little below where the facial canal makes its appearance on the inner wall is seen the processus cochleariformis, through which passes the tendon of the tensor tympani muscle (Plate I, Fig. 5-B).

Fourth—Below the canalis fallopii, near the posterior wall, is a recess at the bottom of which is an oval opening, the fenestra ovalis, measuring three millimeters long, and one and one-half millimeters wide, the long axis of which is directed upward and forward (Plate II, Fig. 8-A). In this oval opening rests the footplate of the stapes, which will be discussed later. This ossicle separates the vestibule of the labyrinth from the tympanic cavity. It is through this fenestra that most of the sound waves travel (Plate I, Fig. 3-D).

Fifth—Below this oval opening, helping to form its lower border and extending forward, is an elevation known as the promontory, which represents the first or beginning turn of the

cochlea, and forms the greater part of the inner wall of the tympanic cavity (Plate II, Fig. 12-D). If we trace this prominence backward, we find it ends abruptly in a deep recess, the recessus rotundus. Opening forward from this recess in the macerated bone is a round aperture, the foramen rotundum, which leads into the beginning of the cochlea (Plate IV, Fig. 21-A). In the recent state this opening is closed by a membrane, the membrana secundaria, which regulates the fluid waves that have passed through the cochlea. Opinions differ as to whether sounds travel through the fenestra rotunda from the middle ear. According to Gruber, this round window lies nearly parallel to the drum membrane in the fetus at the third or fourth month, while at birth it lies obliquely to the membrane, and as the child advances in years this opening gradually changes until it looks toward the posterior wall. Troltsch is of the opinion that the infantile position of the round fenestra may under certain conditions persist. This round opening varies from one and six-tenths to three millimeters in height and its width from one to three millimeters. The membrane which closes this round opening is somewhat convex toward the interior of the cochlea. It is covered on the cochlea side by the labyrinthine lining, and on the tympanic side by the mucosa of the lining of the middle ear.

The tympanic cavity measures through its horizontal plane ten to thirteen millimeters; its vertical plane from eight to fifteen millimeters. The roof increases in height from the anterior part of the cavity to the posterior, where it continues on as the covering of the antrum. The width of this cavity varies; at the umbo and promontory it is about two millimeters, and from that the width varies up to seven millimeters. The quantity of fluid which this cavity will hold is about ten minims in the recent specimen, and in the dried about twelve minims.

This cavity is divided into three chambers by imaginary lines: the upper or attic, the middle or atrium, and the lower or hypotympanic space (Plate III, Fig. 20-D).

The attic is that portion lying above an imaginary line drawn from the prominence of the horizontal portion of the facial ridge to the short process of the malleus. Some authors draw the line to the lower edge of the external table of the squama or upper edge of Shrapnell's membrane. Kretschman and

Siebenman contend the former should be adhered to, and base their opinion on the clinical fact that it is a common observation to find the membrana flaccida involved when the pathologic process is limited to the attic. The attic lies almost directly above the posterior quadrant of the drum membrane (when the head is held in its horizontal plane) and lower ledge of the outer table of the squama. If a perpendicular line were drawn from the inner wall of the attic downward, it would pass through the drum membrane near its upper edge and strike the floor of the external meatus about three millimeters external to the lowest edge of the drum. If drawn in like manner from the extreme outer part of the outer wall, it would strike the floor of the meatus about seven millimeters from the lowest edge of the drum. To this space Bezold, Troltsch and Siebenman have given the name aditus ad antrum; Schwalbe, cavum epitympanicum; Liedy, attic, and Hartmann, the cupola. In this socalled chamber rests the heads of the malleus and incus and their ligaments (Plate IV, Fig. 23-A). The book of Surgical Anatomy by Davis and the book by MacEwen, describe the attic containing the above structures, but continue to say that the short process of the incus projects into a space called the iter or aditus, and that through this space the attic communicates with the mastoid antrum. They describe the space as being a quarter of an inch long from the posterior extremity of the attic to the antrum, and that it is covered by the tegmen, the inner and lower wall by hard bone for the seventh nerve and external semicircular canal. This chamber is of the greatest importance because of the many structures it contains and because of its close association with the cranial cavity and mastoid antrum. Because of the number of structures and reduplication of mucous membrane, infection of this region is arduous to master on account of the difficulty in establishing drainage (Plate IV, Fig. 25-C). The incus aids us in many cases; because of its position, it acts as a natural drain from this region. The attic is again divided into an internal and an external chamber. The area lying between the malleoincudal articulation and the external table of the squama and Shrapnell's membrane is called the external attic; that part internal to these structures, the internal attic. This external attic is

again subdivided into an upper and lower space. The lower one is known as Prussak's, and is bounded internally by the neck of the malleus, inferiorly by the processus brevis, externally by Shrapnell's membrane, and superiorly by the external ligament of the malleus. This space measures about one and a half millimeters high and two millimeters at its base. According to Politzer, this space opens occasionally into the anterior and posterior pouch of Troltsch. From my observations of work in the attic region, I have come to the conclusion that the number of trabeculæ in the attic varies in many cases, and I think it due to the process of development.

The atrium is that part of the tympanum lying opposite to the drum, below the imaginary floor of the attic and above a plane on a level with the floor of the external auditory meatus. In this chamber are found the handle of the malleus and the long process of the incus, stapes, oval, round window and opening into the eustachian tube.

The hypotympanic space lies below the lowest edge of the drum membrane or below the floor of the external auditory meatus, and contains a variable number of cellular spaces.

The ossicles of the middle ear are three: malleus, incus, and stapes.

The malleus is club shape, measures about nine millimeters in length and weighs about twenty-two milligrammes. It is made up of a head, neck and handle. The head and neck form with the handle an angle of about 150 degrees. The head is irregularly globular in shape, and its upper part lies about one millimeter from the tegmen. It has a free convex surface, and on its inner and posterior surface has an oval depression for articulation with the incus. The neck is a fraction over one millimeter long, and extending forward from its anterior surface is a delicate spicule of bone, the processus gracilis, the remains of Meckel's cartilage. Extending in an obtuse angle from the neck, downward and inward, is the handle, which is prismatic on cross sections, and tapers gradually from just below the neck to the tip, which is turned slightly forward and somewhat flattened. The handle measures five millimeters from short process. At the upper end of the handle is a somewhat rounded point, projecting against the drum, and called the short process. On examining the handle while connected with

the drum, the inner two-thirds of its circumference seems to be free toward the tympanicum, excepting for the mucous membrane covering, while the outer one-third seems imbedded in the membrane (Plate III, Fig. 20-B). The membrane can be easily separated from the malleus with the exception of its lower end, where it is firmly attached. Gruber contends that a cartilaginous structure surrounds the malleus where it is imbedded in the drum, and the bony part rests in this cartilaginous bed. This ossicle, especially the head and short process, is practically always preserved in suppurative conditions, because of its rich blood supply, which will be spoken of later. A case of congenital gap in the handle of the malleus has been reported by A. H. Cheatle. The ligaments which hold this ossicle in place are: the suspensory, a short bundle of fibers attached to the upper surface of the head and tegmen tympani; it checks the outward movements of the handle. The external is a fan-shaped structure attached to the neck of the malleus and diverging as it passes outward to become attached to the lower edge of the outer wall of the squama at the Rivinian notch. This ligament forms the roof of Prussak's space. There is a small bundle of fibers at the posterior part of this ligament which is separated from the main bundle, and called by some authors the posterior ligament of the malleus. The anterior ligament leaves the neck of the malleus around the processus gracilis, encircling this process and passing to the Glaserian fissure and anterior spine of the tympanic ring. The capsular ligament surrounds the malleoincudal articulation, and holds these surfaces together. Because of the number of ligaments which are attached to this ossicle, it makes it firm and secures it in one place, therefore, making it a valuable landmark.

The incus, called so because of its shape, has a body, a long and short process. On the anterior surface of the body is a saddle shaped depression, for the articulation of the head of the malleus. The two surfaces of the malleus and incus are so arranged that when the drum membrane is pushed inward by a sound wave, a small ridge on the malleus fits into a depression on the incus, thereby bringing pressure on the stirrup, while if the drum is pushed outward by inflating, etc., these two surfaces separate and the malleus moves outward without

pulling the incus with it, thus preventing the dislocation of the stapes from the oval window during inflation, etc. The short process measures three millimeters, and in some cases a fraction over; the long process, from the lower edge of the articular surface to the tip, about four and a half millimeters. This ossicle weighs about twenty-four milligrammes. The short process is directed backward and is lodged in the sella incudis of the posterior tympanic wall, just below the opening into the antrum mastoideum. The long process, downward and inward, parallel and posterior to the malleus handle. At its lower end it turns at a right angle inward to join the head of the stapes. The right angle projection is known as the processus lenticularis or orbicularis. In the fetus there is a separate bone (Quain). In inflammatory conditions of the tympanum the incus is the first of the ossicles to be thrown off and destroyed because of its limited blood supply, and because of the position of the artery which supplies it, which is so readily pressed upon by the swelling of the tissues in that area. The ligaments which secure this ossicle in position are: the capsular, already spoken of; the ligament of the incus which surrounds the short process, passes back and is fastened to the area of the floor of the iter where the short process is lodged. There is a small ligament which holds the os orbicularis and head of the stapes together.

The stapes consists of a head, two crura and a footplate. The head is directed outward and articulates with the processus lenticularis of the incus. It is disputed as to whether this is a synovial joint, but Rudinger describes it as such. There is a short neck which divides into two crura, the anterior and posterior, which are united to a flat oval plate, the footplate. The footplate occupies the greater part of the fenestra ovalis, but it is in no place in actual contact with the bone, there being a layer of cartilage all around the footplate, and another around the edge of the oval window. Between these two layers of cartilage is a ligament. The length of the footplate is a fraction over three millimeters, its width one and a half millimeters. The anterior crus measures about two millimeters, its posterior crus three millimeters. This ossicle weighs about two milligrammes. The ligaments are the annular, which holds the footplate in the oval window, and one which unites the head with the incus.

The muscles of tympanum are two: the tensor tympani and the stapedius. The tensor tympani arises from the cartilaginous portion of the eustachian tube, small area of the great wing of the sphenoid around the tube, and from the walls of the bony canal which lodges the muscle. It is a little over half an inch long. Near the opening of this tube the muscle becomes tendinous, passes through the processus cochleariformis at right angles to its belly, passes outward across the tympanum to be inserted into the inner surface of the manubrium of the malleus, where it joins the neck. Gruber says the tendon is attached to the inner margin and anterior surface of the malleus.

The stapedius muscle arises from a canal in the posterior wall of the tympanum, lying parallel with the fallopian canal, but a little anterior and internal to it. It emerges through a small opening, the pyramid, to be inserted into the head of the stapes. Rudinger says it is inserted into the lower extremity of the descending process of the incus. In most of the cases which I examined the muscle seemed to vary in its insertion; sometimes it would be attached to the posterior crus, again to the head and posterior crus, then again some of the fibers seemed to extend from the head to the os orbicularis, but most often to the neck.

Various theories are advanced regarding the function of these muscles, but they are beyond the scope of this paper.

The arterial supply to the tympanum is the tympanic branch of the internal maxillary, which enters this cavity by way of the Glaserian fissure; it supplies the anterior part of the cavity, and its branches anastomose along the periphery of the drum membrane with the stylomastoid branch of the posterior auricular. This tympanic branch sends branches into the external auditory meatus, and is called the deep auricular.

The tympanic branch of the internal carotid passes through the anterior wall of the tympanum into the middle ear. The stylomastoid branch of the posterior auricular passes up the fallopian canal with the facial nerve and sends branches through the posterior tympanic wall to the tympanum and drum membrane.

The petrosal branch of the middle meningeal, which anastomoses with the stylomastoid in the fallopian canal, sends

branches into the tympanum. Zuckerkandl mentions a small branch of the stylomastoid artery, which he found constantly passing out from the middle part of the fallopian canal to supply the stapes and membrana obturatoria, then to the promontory to anastomose with the above branches, which he calls arteria stapedia.

The tympanic branch of the ascending pharyngeal, Gray says, ascends through the eustachian tube, while Deaver speaks of its entrance through the tympanic canaliculus with Jacobson's nerve. All these branches which supply the tympanum anastomose freely. However, Prussak states that the arteries of the tympanic cavity have an extensive course without anastomosing with one another. He says they divide at an acute angle into branches the caliber of which are large in comparison to the parent trunk, these branches passing almost at once into small veins, capillaries in many parts being absent.

Because of the free passage of the arterial supply through the periosteum to the subjacent bone, inflammatory processes of this cavity have a great tendency to involvement of the osseous wall.

There is considerable controversy as to whether the arteries of the middle ear anastomose with the arteries of the labyrinth. Politzer describes such branches existing. Shambaugh, who has studied this phase of the subject very thoroughly, has found that there is an anastomosis existing between these two chambers. (*Archives of Otology,* 1906.)

The venous blood of the tympanum is carried off by the temporomaxillary vein, superior petrosal sinus, lateral sinus, internal jugular, and pharyngeal veins.

Numerous small vessels communicate with the dura through the tegmen tympani. Inflammatory processes may travel by these veins to the various areas through which the veins travel and are, therefore, of pathologic importance.

The lymphatic distribution of the tympanum is not well known, but according to Rauber they accompany the blood vessels. There is a close connection with the prevertebral glands, which should be remembered in a retropharyngeal abscess, during middle ear suppuration.

The nerves of the tympanum are derived from several sources. The relation of the seventh nerve to the tympanum

has already been considered. The nerve supply of the middle ear is derived from branches of the fifth, seventh and ninth cranial nerves, which form a plexus on the promontory known as the tympanic plexus. The branches which form this plexus are Jacobson's nerve from the ninth, which passes through the canaliculus of the floor of the tympanum just below the promontory. The small deep petrosal from the carotid plexus of the sympathetic, which enters through the anterior wall. The small superficial petrosal branch of the seventh, which enters the tympanum beneath the tensor tympani canal. The great superficial petrosal, another branch of the seventh, which enters the tympanum just anterior to the oval window.

The chorda tympani nerve passes through the tympanic cavity, entering at the upper posterior arm of the annulus through a small opening, the iter chordæ posterior, passes forward between the long process of the incus and neck of the malleus, just above the junction of the tensor tympani muscle with the malleus, and passes out of the cavity through a small opening at the upper anterior arm of the annulus, the iter chordæ anterior (Plate II, Fig. 12-C, and Plate IV, Fig. 22-A). The position of this nerve in the tympanum causes two pockets to be formed, one anterior and the other posterior to the malleus, and known as the anterior and posterior pouches of von Troltsch (Plate IV, Fig. 24). The mucous membrane passes down from the roof of the tympanum, covers over the chorda tympani nerve, then passes up again to be reflected into the drum membrane; therefore, these pouches open downward and lie between this fold of mucous membrane which covers the chorda tympani and the drum membrane. The posterior pouch is the larger of the two. Troltsch considers these folds of mucous membrane as true duplicatures of the membrana tympani, and has found fibers in them similar to those in the drum. Gruber, after the most careful study, has never been able to demonstrate the fibers of the membrana propria, therefore, he considers them folds of mucous membrane. Rudinger regards them as forming bands of attachment for the upper part of the malleus.

Politzer says there are openings in some cases into these pockets from Prussak's space. I have followed his method of experimenting by putting quicksilver into the pouches and

opening Prussak's space in eight cases, but the quicksilver did not leave the sacs. I also examined these pouches under high magnifying power, but could see no such openings.

The nerve supply of the drum membrane has been carefully studied by J. Gordon Wilson, who says: "The drum of man is chiefly supplied by nerves which enter from the external auditory meatus. They pass in as one large trunk at the upper posterior part of the auditory canal. Numerous smaller branches enter from the periphery. There are but few nerves entering this structure from the tympanum. These branches are derived from the auricular temporalis and vagus."

I am greatly indebted to Dr. Joseph Beck for many suggestions which were of value to me in the presentation of this subject.

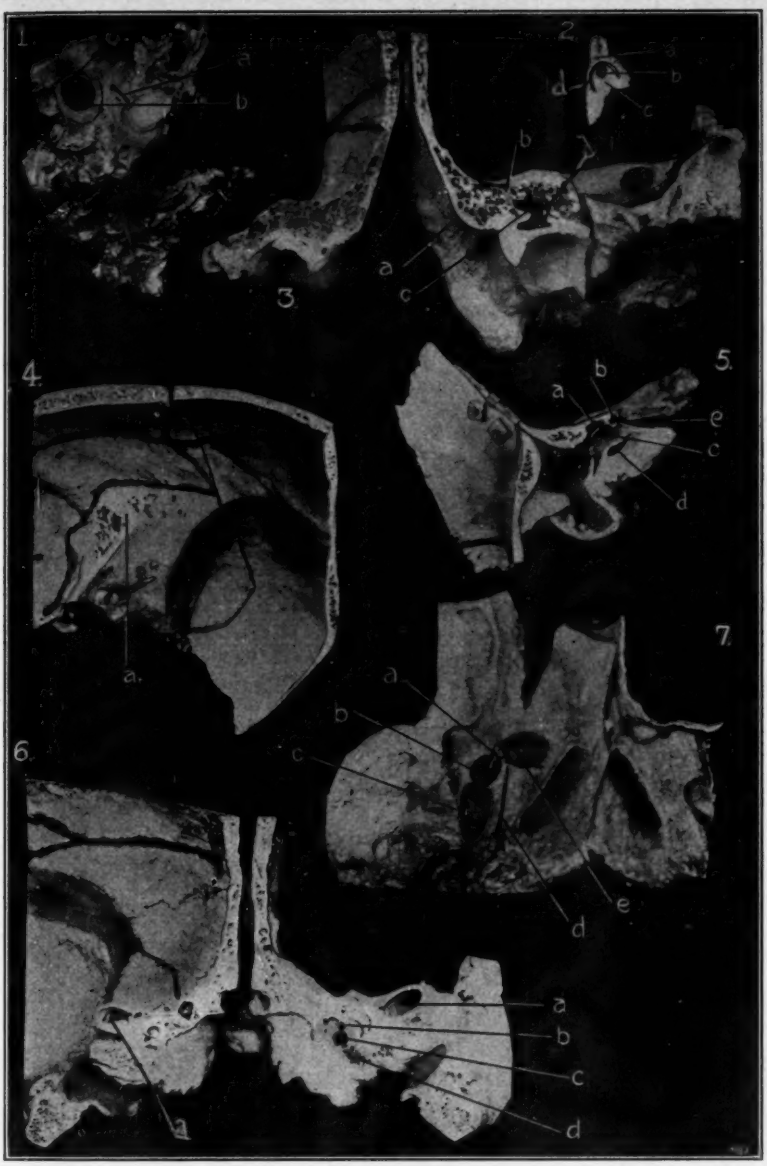


PLATE I.

FIGURE 1.—a—Facial nerve. b—Annulus tympanicus. c—Notch of Rivini.

FIGURE 2.—a—Inward growth of the annulus to help form the floor of the middle ear. b—Foramen of Hueska. c—Outward growth of the annulus to form the external auditory meatus. d—Sulcus tympanicus.

FIGURE 3.—a—External table of the squama. b—Internal table of the squama. c—External semicircular canal. d—Stirrup in the oval window.

FIGURE 4.—a—Dehiscences in the tegmen tympani.

FIGURE 5.—a—External semicircular canal. b—Processus cochleariformis. c—Oval window. d—Lower half of the drum membrane. e—Tensor tympani muscle lying over eustachian tube.

FIGURE 6.—Large cell in the petrous portion of the temporal bone. b—Tensor tympani muscle entering middle ear. c—Eustachian tube opening of the middle ear. d—Cells around tubal opening and in floor of the middle ear.

FIGURE 7.—a—Incus. b—Drum membrane. c—Jugular bulb. d—Facial nerve. e—Mastoid antrum.

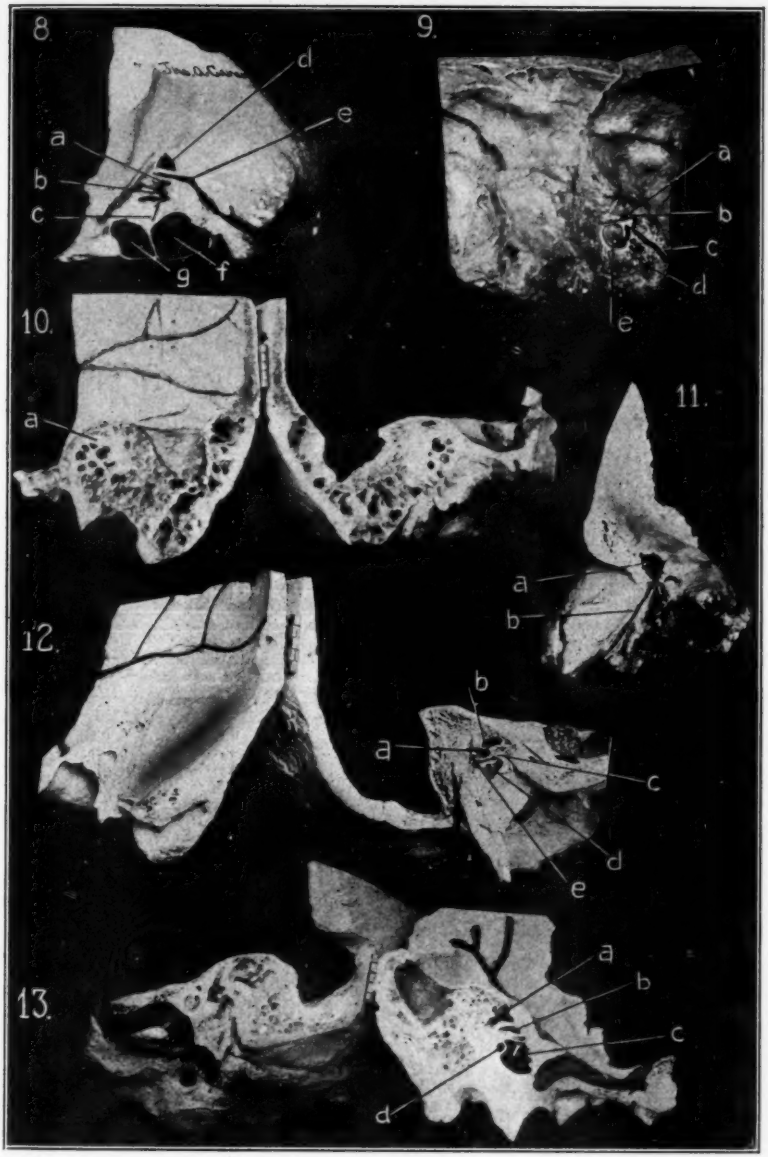


PLATE II.

FIGURE 8—a—Oval window. b—Promontory. c—Jaccbson's or tympanic nerve. d—Opening into mastoid antrum. e—Facial nerve. f—Jugular bulb. g—Internal carotid artery.

FIGURE 9.—a—Chorda tympani nerve. b—Attic. c—Eustachian tube. d—Koerner cells around tubal opening. e—Drum membrane.

FIGURE 10—a—Inner ear.

FIGURE 11.—a—Mastoid antrum. b—Facial nerve.

FIGURE 12—a—Oval window. b—Facial nerve in inner wall of middle ear. c—Anterior arm of the annulus. d—Chorda tympani nerve. e—Posterior arm of the annulus.

FIGURE 13.—View of the middle ear from the inner side. a—Mastoid antrum. b—External semicircular canal. c—Drum and long process of the incus. d—Space in the bone for the stapedius muscle.

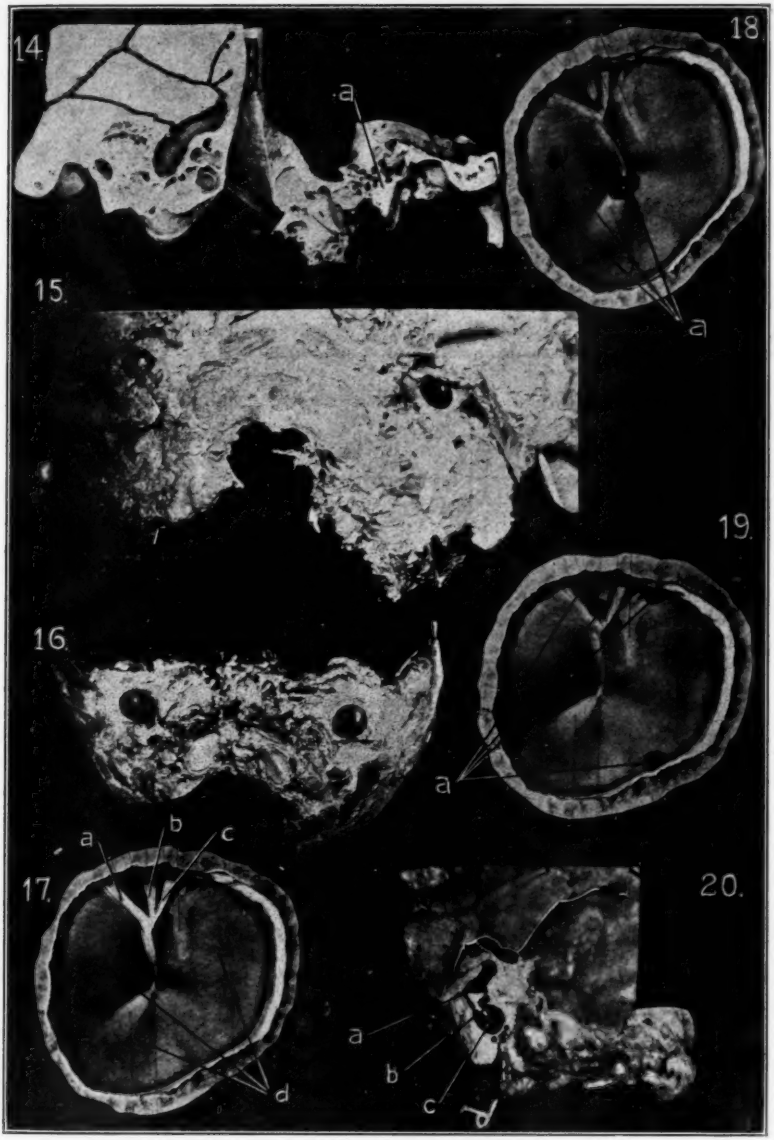


PLATE III.

FIGURE 14—a—Cells around the styloid process.

FIGURE 15.—The adult drum membrane, with the bony and cartilaginous parts of the meatus removed as far as the annulus.

FIGURE 16.—Infant drum membrane and annulus at birth.

FIGURE 17.—(Deaver.) a—Anterior fold or anterior liminal strand. b—Shrapnell's membrane or membrana flacida. c—Posterior fold or posterior liminal strand. d—Membrana tensa.

FIGURE 18—a—The nondangerous variety of perforations of the drum membrane.

FIGURE 19—a—The dangerous variety of perforations of the drum membrane.

FIGURE 20.—a—The attic leading to the antrum. b—The incus. c—Drum membrane. d—Hypotympanic space.

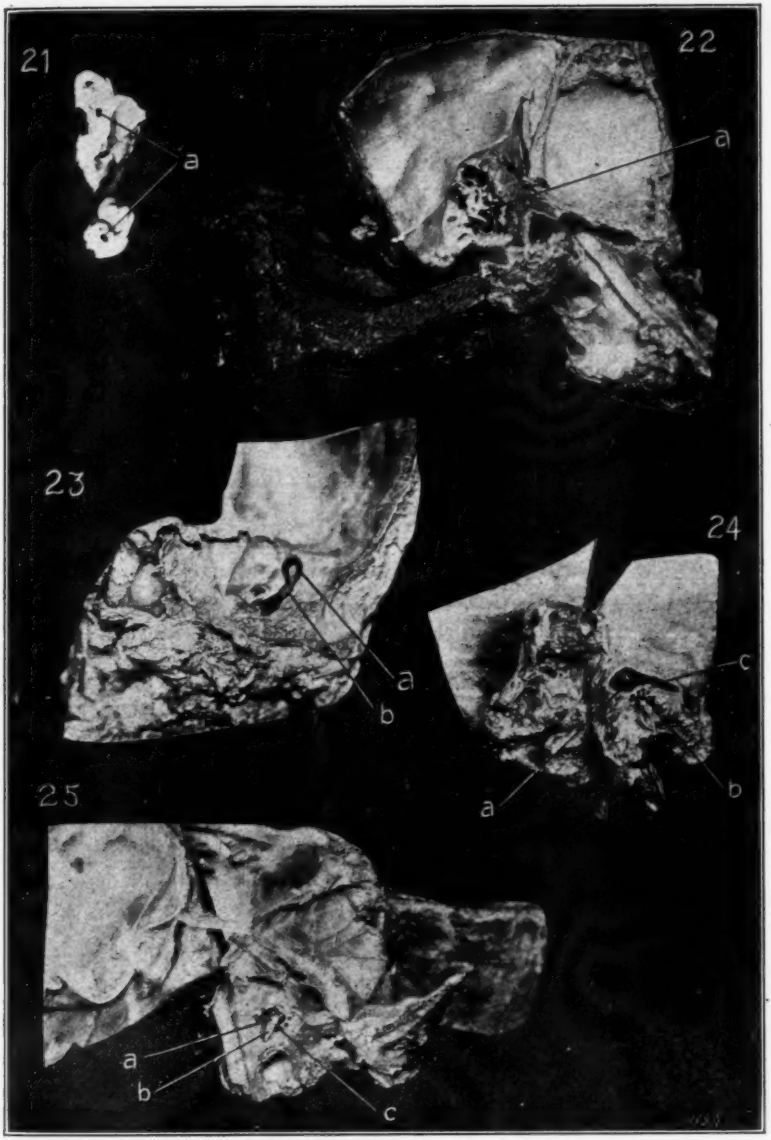


PLATE IV.

FIGURE 21.—a—The round windows.

FIGURE 22.—Outer wall of the middle ear, viewed from within; showing opening into the antrum, attic, ossicles, and drum membrane.

FIGURE 23.—a—Attic. b—Atrium. Malleus in position.

FIGURE 24.—a—Posterior pouch of Troltsch. b—Koerner cells around opening of eustachian tube. c—Eustachian tube.

FIGURE 25.—Middle ear from above, with the tegmen tympani and antri removed. a—Head of malleus. b—Short process of the incus. c—Fibrous bands between heads of the ossicles and external table of the squama.

XLVIII.

SOME ANATOMIC AND CLINICAL RELATIONS OF THE SPHENOID SINUS TO THE CAVERNOUS SINUS AND THE THIRD, FOURTH, FIFTH, SIXTH AND VIDIAN NERVES.*

By Greenfield Sluder, M. D.,

St. Louis.

The body of the sphenoid bone is usually hollowed out by the sphenoidal sinus. This cell may, however (rarely), be rudimentary and occupy a very small space in the lower anterior part of the body, which is otherwise hollowed out by a postethmoidal cell. It is of the cell which hollows the body of the sphenoid that I speak, regardless of whether it be the sphenoidal sinus proper or a postethmoidal cell.

The body of the sphenoid is covered above and laterally by the dura mater, with the cavernous sinus between its external and internal surfaces (in it), occupying a position for the most part above and lateral to the body. Within the cavernous sinus are found the internal carotid artery and the third, fourth and sixth cranial nerves, with the first division of the fifth lying in the lower part of its lateral wall. The impression given in the treatises on anatomy is, usually, that these nerves are rather widely separated from the sphenoid sinus, as shown in Figure 1. The second and third divisions of the fifth and the Vidian are also usually represented as well removed from this cell—that is, separated by a considerable thickness of bone. (Fig. 1.) The fact is, the sixth and the third division of the fifth are the only ones of these nerves that are not at times in close association with this cell—that is, separated from it by a very thin layer of bone; and even the third division of the fifth is sometimes also in rather close

*Read before the American Laryngological Association, Atlantic City, N. J., May 11, 1912.

association with it. (Figs. 2i, 2ii.) The sixth, so far as I have found, is uniformly placed on the lateral aspect of the carotid and always removed from this bony wall. Thus far I have been speaking of a sphenoid sinus which is limited to the body of the sphenoid. Should it be extended into the greater wings, the sixth nerve may come into close association with it in the sphenoidal fissure below.

The fact which determines the relations of these nerve trunks to the sphenoid sinus is the size of the cavernous sinus rather more than the size of the sphenoid sinus. If the cavernous sinus be large in length and cross section, these nerve trunks will usually be far removed from contact with a sphenoid sinus of any size. On the other hand, if the cavernous sinus be small in length or cross section, these nerve trunks may be closely associated with a sphenoid sinus limited to the body of the sphenoid bone. Of course, a small sphenoid sinus in the center of the body of the sphenoid bone will be widely removed from contact with these nerve trunks, regardless of whether the cavernous sinus be large or small. A sphenoid sinus of large extent—prolonged backward and outward—may readily closely approach the third division of the fifth in the foramen ovale (Figs. 2i, 2ii), and Dr. H. P. Mosher has shown me two specimens in which the sinus extended to a close association with the Gasserian ganglion. In these two specimens the internal carotid rising out of the foramen lacerum medium was surrounded on the internal half of its circumference by the sphenoidal sinus, the wall of which was less than the thickness of a hen's egg shell at this place (Fig. 4ii.) A sinus extended downward soon approaches the Vidian nerve. In fact, the upper side of the bony case of the Vidian canal is often partly deficient under these circumstances. (Figs. 2ii, 3, 4.) The excavation may also extend below the level of the canal and leave it stalking through the sphenoid sinus like an ancient aqueduct, over the plain, connected with the bone below by a paper-like support. (Fig. 3.) Or it may be protected on one side of, and exposed on the floor of the sinus of the other side of the skull. (Fig. 4.) The sphenoid sinus extended laterally soon reaches the foramen rotundum, and may then environ one-half of its circumference, bringing the second division of the fifth at this point,

and for some distance posterior to it, into very close association with it. (Fig. 5.) The third and fourth nerves are in close association with the anterior clinoid process or lesser wing of the sphenoid, which are not infrequently hollowed out by a prolongation of the sphenoid sinus or (from in front) by an extension from a postethmoidal cell, thereby bringing these nerves also into close association with these cells. (Fig. 6.) The first division of the fifth comes into close association with the sphenoid sinus anteriorly, if the cavernous sinus be small in either direction. (Figs. 7, 8.)

Clinically the inflammatory diseases of the accessory sinuses of the nose vary greatly. Each has, however, some semblance of constancy in its behavior, at least as far as symptoms with signs go, with the exception of the sphenoid. As is known to us, it is no uncommon thing to see a sphenoidal empyema give rise not only to its own characteristic symptoms, but to simulate the pains produced by all the other sinuses. Pain of a frontal sinus inflammation may be simulated by a sphenoidal inflammation—the differential diagnosis being made by the absence of pus from the frontal, with no tenderness of its floor, as well as a negative finding on the Roentgen ray picture. The pain of a maxillary antrum inflammation may be differentiated by a negative antrum puncture as well as a negative Roentgen ray picture. Absence of pus in or from the ethmoid would exclude the ethmoidal cells. These symptoms, when produced by the sphenoid, are, however, more apt to be nocturnal than diurnal.

It seems to me there are two pictures produced by inflammatory disease of the sphenoid sinus. The primary simple one is that produced by the pressure from an obstructed outflow of pus. I have construed this as the explanation of the somewhat "dull," "heavy" pain in the back of the head described by these patients. The more complex one, simulating the pains which are ordinarily produced by the other sinus, can only be explained by assuming that the associated nerve trunks have become involved either by the inflammatory process or by its toxins. This assumption applied to the third nerve explains also the dilated pupil seen in some of these cases, when the ophthalmologist or neurologist can assign no reason for it; and the asthenopia that is sometimes met with when

no reason may be found in the eye or nose, otherwise. Some of these cases show a paresis of the superior oblique.

From the above sketched anatomy it would seem possible with the arrangements cited, by which the sphenoid sinus is separated from the nerve trunks by thin divisions of bone, that such an extension of inflammation might easily occur. In an effort to prove the accessibility of these nerve trunks I have ten times painted the cavity of the sphenoid in a stripe from above downward and outward with a very small applicator bearing about one-half drop saturated water solution of cocain and paralyzed the three divisions of the fifth for tactile sensation and pain, accompanied by a marked sense of stiffness of the lower jaw on that side. A needle passed through the skin supplied by these nerves failed to evoke sensation. In these cases the separating wall must have been a very thin one, thereby permitting the cocain to pass quickly and easily through to the nerve trunks. I have also, for therapeutic purposes, filled the sphenoid with oil solutions—1 per cent phenol, 2 per cent menthol, 5 per cent and 10 per cent oil wintergreen and 1 per cent cocain alkaloid—and found that they all produce a well defined analgesia of the first and second and often the third division of the fifth, accompanied by very little or no tactile anesthesia. It therefore seems to me reasonable to assume, when the diagnosis is sphenoid empyema and the symptom is pain in the brow, that it is inflammation or irritation of the first division of the fifth by the process within the sinus which makes the symptom. When the symptom is pain in the upper jaw and teeth, it seems reasonable to assume the second division of the fifth has become involved. When the symptom is pain in the lower jaw and teeth, accompanied by a sense of stiffness in the jaw on that side, it may be assumed that the third division has become involved and that probably in the foramen ovale.

The behavior of certain sphenoid empyemata has interested me much for several (five) years. These are cases in which the pains and aches have continued unchanged, despite wide opening of the cavities and cessation of all pus or signs of local disease. It is my habit to perform the Hajek radical postethmoidal sphenoidal operation. It seems to me the most comprehensive treatment. Some of these cases, however,

although recovering from the suppurative inflammation, and later from all signs and symptoms, have frequently subsequently developed headache and behaved as cases of migrain. Dr. M. A. Bliss has seen a number of these cases in consultation, and has sent some of them to me and has agreed in the diagnosis of migrain. We have felt that in at least this class of cases which have simulated and borne the name of migrain, that the origin of the pain was a local one. These are of similar symptom complex to those of toxic origin. The distribution of the pain has been that suggesting as its origin the first division of the fifth, as, or well as, the second division, sometimes, or the third division, and combined at times with the posterior pain, which I have attributed to the irritation of the Vidian. In a recent paper[1] I mentioned that in the injection of the sphenopalatine ganglion with alcohol, when the needle was placed too far internal, the pain produced by the alcohol was projected backward and into the shoulder, etc., and when placed too far external the pain was referred to the upper jaw—that is, when the side of the ganglion upon which the Vidian enters it was injected the pain was referred posteriorly, whereas when the side upon which the second division entered it the pain was referred to the upper jaw. When the alcohol was placed in the ganglion direct the pain was referred in both directions. (The relative position of the Vidian and second division of the fifth is shown in Fig. 9, which portrays the foramina through which they pass at these points.)

These cases of recent origin (six months to two years approximately) when well of the suppuration were usually well of all symptoms and remained so until a coryza infected the general nasal cavity, when they again developed their pains, often without suppuration of the sphenoidal and postethmoidal sinuses. The membrane of the sphenoidal sinus, however, usually showed under these circumstances a swollen, edematous condition (even polypoid) analagous to that shown by the anterior ethmoidal region in hyperplastic ethmoiditis under similar inflamamtory influences. In the recovery the swelling and edema would subside, but more or less of the pain would continue, and at a time later begin a cyclical reappearance, with no visible disturbance in the sphenoid sinus, even

when its anterior wall had a permanent opening in it large enough to permit a good view of its interior. I have observed a similar behavior of the sphenopalatine ganglion, i. e., intermittent reappearance of the neuralgia. I have also observed in these cases a picture identical with the neuralgia which starts in the sphenopalatine ganglion. This is not surprising —on the contrary, would be expected when one proves the accessibility of these nerve trunks. The entire nerve supply of that ganglion—namely, the second division of the fifth and the Vidian nerve—being so closely associated with the sphenoid sinus, the picture of the ganglion neuralgia is readily reproduced under inflammatory conditions within the sinus. The differential diagnosis, it seems to me, is made by the facts —that cocainization of the ganglion stops the pain when it is originated in the ganglion, but fails to stop it when it is originated in the sphenoid, because the ganglion is peripheral to that point of origin; and that cocain solution applied within the sphenoid sinus does stop the pain. In the case of ganglion neuralgia, the membrane covering the sphenopalatine foramen is often, but not always, congested. This appearance is absent in the case where the pain is started in the nerve trunks from within the sinus.

I have seen this class of sphenoidal cases behave as vasomotor rhinitis or rhinorrhea or "hay fever," as the patients call that form of paroxysmal sneezing, accompanied by profuse watery discharge and by asthma. In these cases all therapeutic measures applied peripherally (intranasal) were of no avail. The cases yielded to intrasphenoidal applications. The one so far most usually acceptable has been a 2 per cent to 5 per cent solution of sodium salicylate.

The history of one of these cases bearing on this phase of their behavior I herewith relate:

Case 1.—Miss M. M., thirty-seven years old, consulted me on February 26, 1908, for general headache with great morning sneezing. There was swelling of the cavernous tissues in the nose with profuse general watery secretion; but no pus or visible evidence of sphenoidal inflammation. In the effort to control this syndrome the anterior nasal nerves were injected with alcohol (Stein) and trichloracetic acid applied to the tubercle of the septum (Francis) and middle turbinate,

finally the galvanocautery to lower turbinates and tubercle, November 6, 1908; December 26, 1908. The nose was opened for air, but the sneezing continued. All efforts failed. She had to abandon a busy and very useful life for an indefinite period of rest. Dr. D. B. Delavan, who saw her in consultation, advised rest in the mountains. After six months in the mountains of the South she returned to St. Louis, well, and remained so for one year. At this time, March 17, 1910, she returned for treatment because of a "bad cold and headache," as she expressed it. She showed a bilateral postethmoidal sphenoidal suppuration, for which I eventually operated, both sides (April 21, October 27, 1910). On January 10, 1911, she returned to me for an acute coryza with general headache and almost insufferable sneezing (sneezing to exhaustion), without evidences of postethmoidal sphenoidal suppuration, for which she was again compelled to retire to the mountains. June 12, 1911, she again reported to me well. October 12, 1911, she consulted me because of intractable sneezing and general headache with intense burning, stinging sensation in the nose ensuing upon a coryza. This time I began the intrasphenoidal application of 1 per cent cocain in oil, with 1 per cent phenol in oil, which controlled the sneezing and headache fairly well. A little later I began applications of 2 to 5 per cent sodium salicylate water solutions, which proved much more satisfactory—the sneezing and headache stopped in about six days. February 17, 1912, a coryza again brought on general headache and sneezing. This time, knowing that salicylate of methyl was a more potent remedy than the soda salt, and that it was usually tolerated, I filled the sphenoid with a 5 per cent solution synthetic methyl salicylate solution in oil. To my surprise and disappointment, it aggravated the entire syndrome in great severity, and in addition produced asthma lasting five days. A little later, after applications of the sodium salt, she became comfortable; and this in a much shorter time than in the preceding attacks. Locally this case behaves like a hyperplastic ethmoiditis. In the inflammatory stage the membrane becomes edematous with polyp formations, which subside when the attack is over.

That postethmoidal sphenoidal inflammation may give rise to a periodic headache, which is usually spoken of as migrain, is illustrated by the following history:

Case 2.—J. H. A., thirty-seven years old, strong, healthy man, had never had a headache in his life that he remembered; consulted me May 27, 1910, because of a severe left-sided headache which had lasted three weeks. He showed a left sphenoidal postethmoidal suppuration. His pain was parietal and occipital, for the most part, and irregular in severity, sometimes stopping. When severe it was combined with pain in brow and upper jaw. He was melancholic. Treatment was not satisfactory. (He was compelled to be out of the city five days of every week.) On October 12, 1910, he took the proposed operation. The reaction was severe. He was free of symptoms in three months. March 4, 1911, he consulted me because of a coryza with pus which recovered spontaneously. January 6, 1912, he returned because of a coryza with headache, which did not recover spontaneously. At that time he did not show pus in the sphenoid. He had suffered greatly from intermittent general left side headache. Believing that the anesthetic qualities of carbolic acid could be soaked into the nerve trunks in juxtaposition to the sphenoid, I filled the cavity with 1 per cent solution in oil, which stopped the pain in six hours. The cavity was filled once a week for three weeks, when he seemed well, and remained so until the next coryza, six weeks later, which was controlled by 1 per cent carbolic acid in oil. The left pupil is larger than the right during his attacks, and use of his eye is somewhat difficult at those times. He says his pupils were always equal before his headache began and are so now in the intervals between coryzas when he is well of headache. This case has so far not behaved as a hyperplastic ethmoiditis. In the inflammatory attack it shows only redness with little swelling, and sometimes pus.

Case 3.—Mrs. Buc., aged forty years, consulted the Nose and Throat Clinic, O'Fallon Dispensary, Washington University Medical Department, for severe headache, worse on left side, extending to neck and shoulders. All examination was negative. After considerable effort I determined a postethmoidal sphenoidal inflammation right and left, and later, February-July, 1910, opened both sides. The reaction was severe. After some months she was better, and finally became comfortable. A coryza without suppuration later reestablished

the pain, which on the right side was stopped by intrasphenoidal applications of 10 per cent salicylate of methyl, and much improved on the left side. Applications of cocain inside the left sinus quickly produced total anesthesia and analgesia of the areas supplied by all the divisions of the fifth nerve, with a marked sense of stiffness of the lower jaw of that side, which I took to be the effect of the cocain upon the motor function in the third division. Dr. D. E. Jackson, of the Pharmacological Department, Washington University, agrees with me in this conclusion. The right side ceased to be painful six months ago, which is shortly after the applications of methyl salicylate were begun. The left continues to be intermittently painful to a considerable degree, but is markedly helped by methyl salicylate, and remains vastly better than before this treatment was begun. She frequently complains of stiffness of the lower jaw on that side. Only once did this case show a tendency toward edma with polyp formation at the time of acute inflammatory attack, and then only slight.

Case 4.—Miss A. McM., thirty-eight years old, consulted me March 20, 1911, because of intermittent headache from which she had suffered for years. At this time it had been coming much oftener and has been rapidly growing more severe, then requiring 2½ grains of morphin sulphate subcutaneously administered by her physician to control the pain. She never took morphin herself. The pain was worse in left temple; but in the height of the attack it seemed to extend over the entire head. And very rarely it came on the right side alone or would be much worse on that side. A postethmoidal sphenoidal inflammation was found on the left side, which was operated two months later. The reaction was intense. She improved little or none during the next four months under a treatment of simple cleansing of the cavities. At this time (October, 1911), intrasphenoidal applications of 10 per cent methyl salicylate were begun with the idea of soaking the medicament into the environing fifth nerve. Since then the general improvement in her suffering has been very considerable. Much of the time she is free of pain, and a large part of the remaining time she suffers only slightly. The recurrence of the intensely severe attacks is less frequent and intense. She recognizes two kinds of pain—one that I can stop by filling

the sphenoid sinus with cocain methyl salicyate or menthol solutions, and another distributed over the same area which I have tried in vain to stop by these measures. When this attack begins it progresses despite all efforts to stop it; but it has become less severe and less frequent under this treatment, and the last attack, April 1, 1912, was on the right side only.

This case has never in any way suggested by its clinical behavior a similar condition to that of hyperplastic ethmoiditis. It has always appeared a simple inflammatory condition of low grade with once a suppurative stage also of low grade. Since the primary appearance disappeared this district of the nose has appeared normal.

Case 5.—Mrs. C. Siek, aged twenty-five years, consulted me February 20, 1903, for an acute pansinus suppuration with general headache. Prior to this attack she had very rarely in her life been sick in any way. She did not remember to have ever had a bad headache, and had always been strong. She has done hard work (shoe machine) since her nineteenth year. Since this attack she has had, up to the present time (nine years), a recurrent headache that seldom skips more than ten days. Much of the time it is frontal, sometimes maxillary, and often intense combined with occipital pain. It is worse at night and during menstruation. Meanwhile I have opened the frontal sinuses and antra and ethmoids and sphenoids, and done everything known to possibly help (contacts, etc.). I have from time to time put her in charge of specialists in all the departments of medicine, who have found her normal, save hyperopic astigmatism, for which she wears glasses. November, 1911, I proved that her trigeminus was easily accessible (intrasphenoidal) on both sides. Since then I have filled the sphenoid once in ten days with 1 per cent phenol in oil. She says that this is the first medication to help her pain.

All characteristics of a nose case disappeared from this patient by the end of the first three months.

I could report another case similar to this, of four years' standing, in which there has been a recurrent sphenoidal suppurative inflammation at long intervals with great exacerbation at these times.

Case 6.—Mrs. M. D. A., forty-eight years old, consulted me

June 11, 1910, because of right side recurrent sphenopalatine ganglion neuralgia, or, at least, what seemed to be such. It had lasted twenty-four years. It was stopped by cocainization of the sphenopalatine ganglion and controlled for one year by carbolization[2] of the ganglion, at the end of which time she suffered some kind of an explosion which took in her entire head, upper and lower part, on both sides, which confined her to her bed for four weeks despite acetphenetidin, aspirin, codein and morphin. I did not see her during this illness. After this attack, however, she came to see me, but showed no local changes. She continued to suffer very greatly and frequently from what seemed, from its distribution, a trigeminal neuralgia with pain in her occiput and neck, almost altogether on the right side. After a careful explanation of her case, describing the local conditions and telling of a possible benefit from intrasphenoidal medication, Dr. Bliss and I advised her to submit to the opening of her sphenoid, despite the fact that no sphenoidal inflammation betrayed itself. She accepted the proposal. Twice prior to the Hajek radical postethmoidal sphenoidal operation I injected a drop of 5 per cent solution carbolic acid[2] in 95 per cent ethyl alcohol into the sphenopalatine ganglion, each being allowed to rest five days with the idea of benumbing it and thereby reducing the reactionary shock. The pain of the operation under cocain and the succeeding depression and pain were, in this case, astoundingly little. Subsequent filling of the sphenoid sinus with 2 per cent sodium salicylate water solution has been accompanied by a decided betterment, both in severity and frequency of the pain on the right side.

Case 7.—Miss M. R., twenty-one years old, consulted me March 24, 1912, because of a mild posterior headache with dilatation of the pupil on the right side, accompanied by discomfort in use of her eyes, of one week's standing. Ophthalmologic examination was negative. An upright rod appeared to her bifurcate below, which, I understand, indicates a paresis of the superior oblique. The anterior wall of the right half of the sphenoid was reddened and bathed in serum. The effort was made to fill the sinus with 1½ per cent sodium salicylate water solution. This was repeated three times in nine days, when she was free of all signs and symptoms, and re-

mained so ten days, when she had a "sick headache" (in which I did not see her) and again had a dilated pupil which became normal when the headache stopped. No discernible cause could be assigned for the sick headache.

The difficulties that beset some of these diagnoses need not be considered by me. They were emphasized by Dr. Casselberry in his paper before this association last year. I have selected a series of cases which pass by graded stages from the most evident to the least evident, if indeed Case 6 be admitted to the company of sphenoidal cases. These cases are not unique. They have been selected from a considerable number. Many times have I seen the same features in other cases, with the exception of Mrs. M. D. A., Case 6, in whom I opened the sphenoid, which appeared normal, in order to permit of intrasphenoidal medication in the effort to soak the remedies through bony walls directly into the nerve trunks. It has been of apparent benefit under extreme circumstances; but the result is not of sufficient time standing to admit of conclusions. In the effort to forestall the shock and subsequent reaction and pain of Hajek's radical postethmoidal sphenoidal operation I have five times carbolized the sphenopalatine ganglion with marked success.

Some of the other cases have had optic atrophy. The vision would diminish under coryzas. Pain lasted much of the time.

In the observation of these cases I am led to the belief that a large number of the frequently recurring headaches, of whatever length of time standing, that usually bear the name "migrain," that are met with in the general practice of medicine, that have defied diagnosis and treatment, are sphenoidal inflammations existent, or were started as such. They may have lost all the evidences of local disease, which some months before were easily recognizable. I believe that the nerve trunks have become diseased from juxtaposition, just as the optic is known to do. Disease of the nerve trunks under these circumstances is in nowise different in its clinical behavior from that which is started further toward the periphery, as, for instance, from an antrum of Highmore suppuration or a diseased tooth, which after the cure of the local disorder continues painful at intervals or becomes a tic-douloureux. The second division of the fifth in the foramen rotundum is in

almost as close association with the sphenoid sinus as is the optic nerve in the optic canal, and under identical setting, namely, surrounded completely by a firm, bony ring. This is true of the third division of the fifth also; but it is not, as a rule, so closely associated with the sphenoid sinus. Optic nerve disease, secondary to the sinus disease, is too well known and proven to require argument from me. I see no reason why we may not assume as much for the other nerve trunks, in close association with these sinuses, with the difference between motor and sensory nerves.

This sphenoidal area being central to the place of injection of alcohol into the branches of the fifth nerve, from under the zygoma, may explain some of the cases that have not been relieved by that treatment. Also, for the same reason can we understand that when the Gasserian ganglion proper has become involved and gives rise to the pain, intrasphenoidal applications will fail to relieve.

The involvement of the nerve trunks in the sphenoidal district producing the sneezing cited in Case 1 explains why the injection of the branches in the neighborhood of the sphenopalatine foramen (Stein), combined with the same treatment of the anterior nerves, has failed to influence the sneezing in some cases, the sphenoidal area being central to the other nerves.

It would appear from Case 2 that whatever may be the lesion of the nerves it is rather easily remediable in its earlier stages by intrasphenoidal medication. How far such patients as cases 5 and 6 may be helped by intrasphenoidal medication remains to be learned.

I have not thus far been able to secure any postmortem material bearing on the above question.

The various medicaments mentioned above have been distinctly advantageous in the treatment of some hitherto irremediable cases (at least in my hands). There is, however, much to be desired—many remedies remain untried. (In many respects this paper must be considered a preliminary report.)

That the cavernous sinuses may become infected and thrombotic, secondary to sphenoidal infection, is well known. In these considerations the cavernous sinus, however, plays only an anatomic part.

I have cited in the records of some of these cases that they behaved as a hyperplastic ethmoiditis, and that some did not. So far as I have been able to learn, there is no postmortem histologic proof of a hyperplastic process in the sphenoid such as has been proven in the ethmoid. This possible condition has been thought to exist and reasoned out by Hajek[3] in his third edition. He also laments the absence of postmortem material.

REFERENCES.

1. Sluder, Greenfield: The Syndrome of Sphenopalatine Ganglion Neurosis. American Journal of the Medical Sciences, December, 1910; also Transactions American Laryngological Association, 1910.

2. Sluder, Greenfield: A Phenol (Carbolic Acid) Injection Treatment for Sphenopalatine Ganglion Neuralgia. Journal of the American Medical Association, December 30, 1911, Vol. LVII., p. 2137.

3. Hajek: Pathologie und Therapie der entzuendlichen Erkrankungen der Nebenhoelen der Nase. Wien (3rd ed.), 1908.

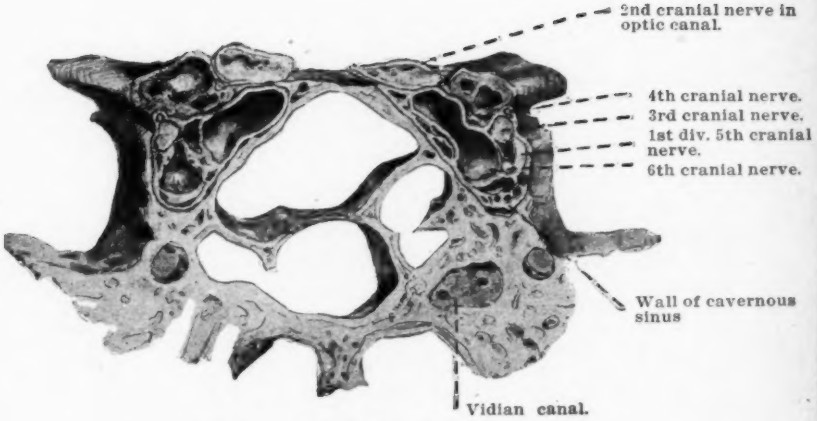


Fig. 1.* (Two diameters.)

Cross section of a cavernous sinus of large length and breadth, at a point far forward, as indicated by the section through the optic nerves and anterior clinoid processes seen from behind, showing all nerve trunks well separated from the sphenoidal sinus. This section shows only the right sphenoidal sinus.

---

*This figure was published in the Archiv fuer Laryngologie, 27 Band, 3 Heft An error occurred in this publication of this (Fig. 1) drawing, in the numbering of the nerve trunks. The 4th was marked 3rd, the 3rd was marked 1st div. of 5th, the 1st division of the 5th was marked 6th, the 6th was marked 2nd division of 5th. And these were all marked "in the optic canal," which, of course, was impossible.

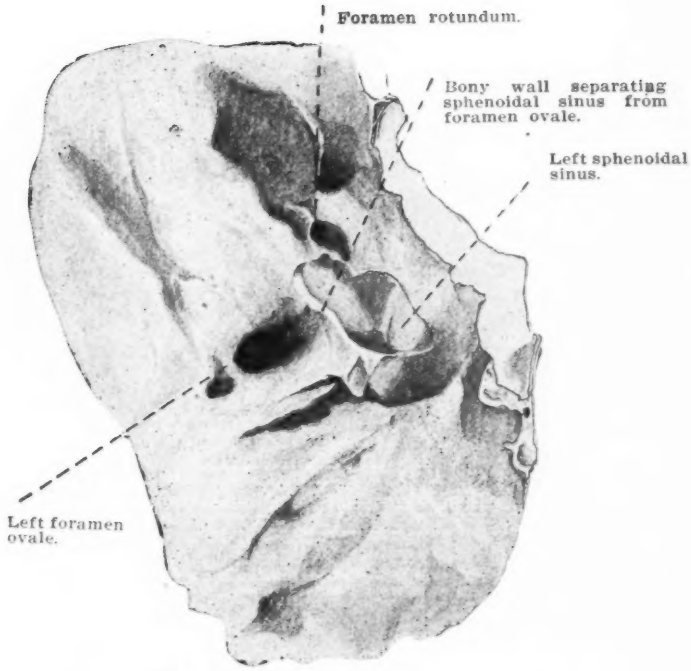


Fig. 21. (Two diameters.)

Left sphenoidal sinus and foramen ovale seen from a point above and to the left.

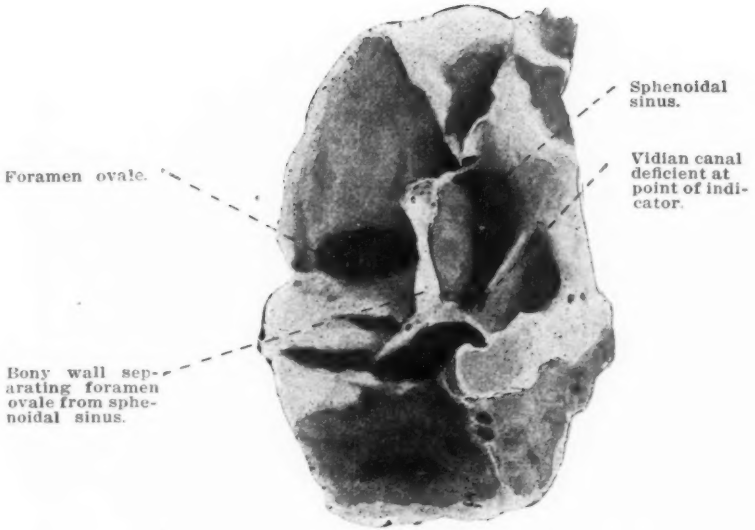

Sphenoidal sinus.
Vidian canal deficient at point of indicator.
Foramen ovale.
Bony wall separating foramen ovale from sphenoidal sinus.

Fig. 2ii. (Two diameters.)

The same specimen as shown in Fig. 2i. Seen from a point above and to the right.

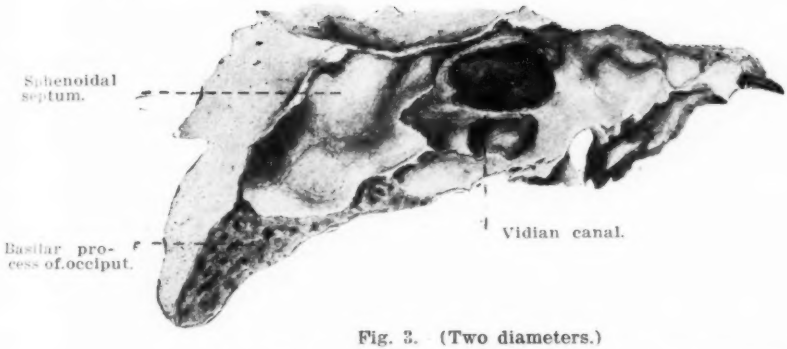


Fig. 3. (Two diameters.)

Interior of left sphenoidal sinus, showing Vidian canal supported by thin bone with the sinus cavity and below it. The canal is open on top.

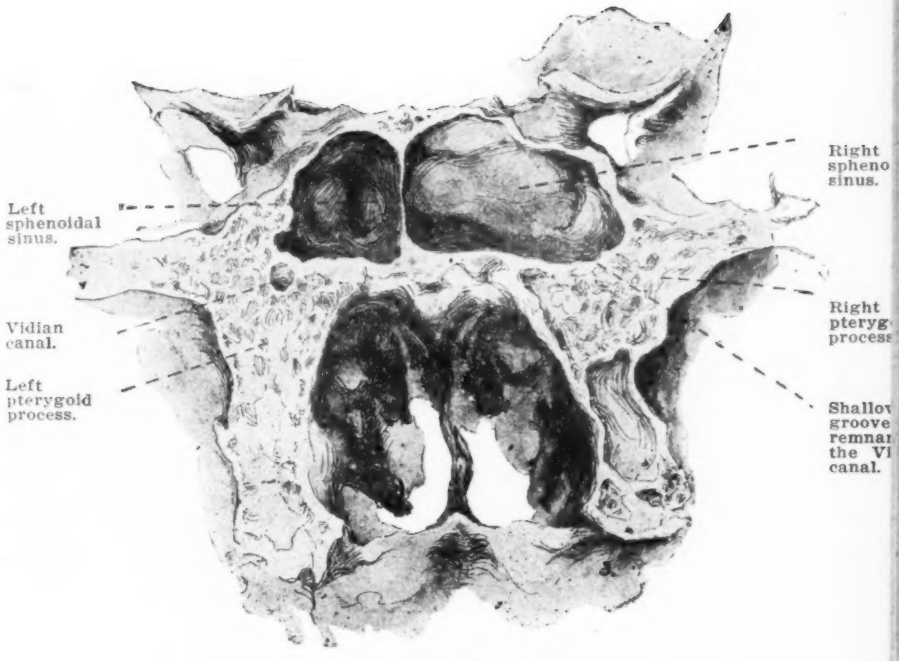


Fig. 4. (Two diameters.)

Cross section of a skull through the pterygoid processes. Seen from behind, showing the Vidian canal of left side well imbedded in the bone; and that of the right side exposed in the sphenoidal sinus.

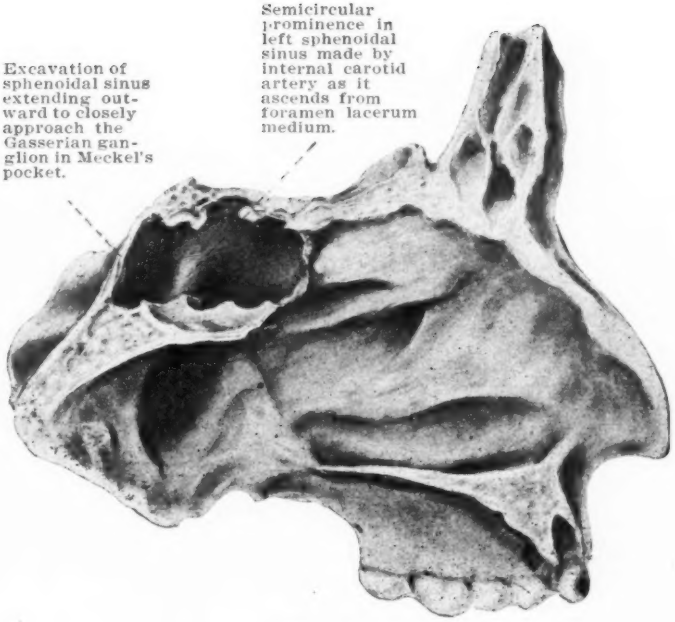

Semicircular prominence in left sphenoidal sinus made by internal carotid artery as it ascends from foramen lacerum medium.
Excavation of sphenoidal sinus extending outward to closely approach the Gasserian ganglion in Meckel's pocket.

Fig. 4ii.

Picture of specimen loaned me by Dr. H. P. Mosher.

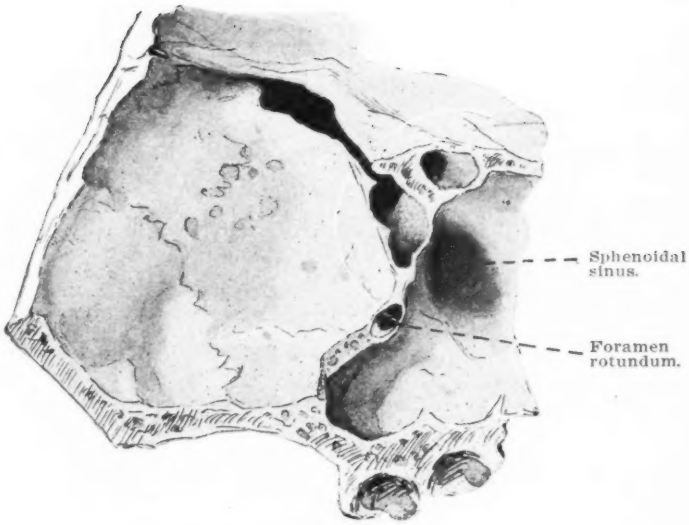


Fig. 5. (Two diameters.)

Middle fossa of skull, showing foramen rotundum separated from sphenoidal sinus by thin film of bone.

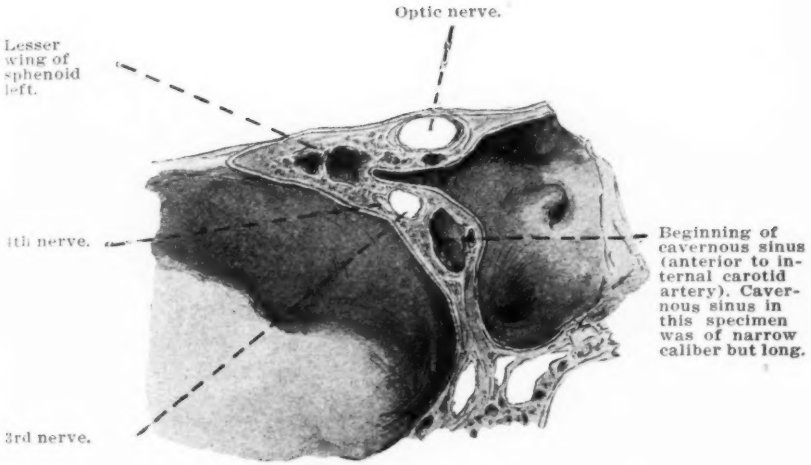

Optic nerve.
Lesser wing of sphenoid left.
4th nerve.
3rd nerve.
Beginning of cavernous sinus (anterior to internal carotid artery). Cavernous sinus in this specimen was of narrow caliber but long.

Fig. 6. (Two diameters.)

Left sphenoidal sinus seen from behind, showing a prolongation into lesser wing of sphenoid, bringing 3rd nerve in close association with it.

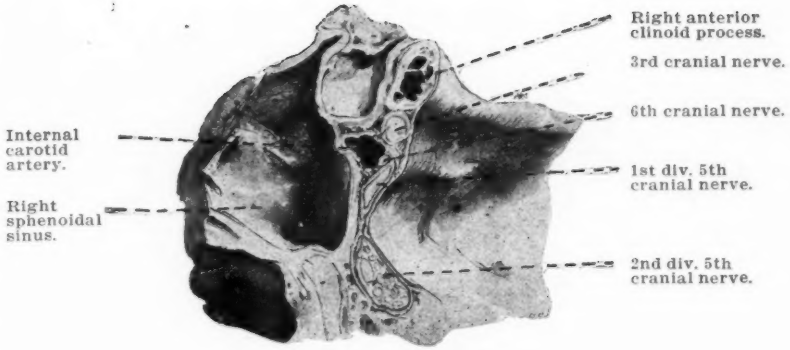


Fig. 7. (Two diameters.)

Cross section of right cavernous sinus (long and narrow), seen from behind, showing first division of 5th nerve in close association with sphenoidal sinus.

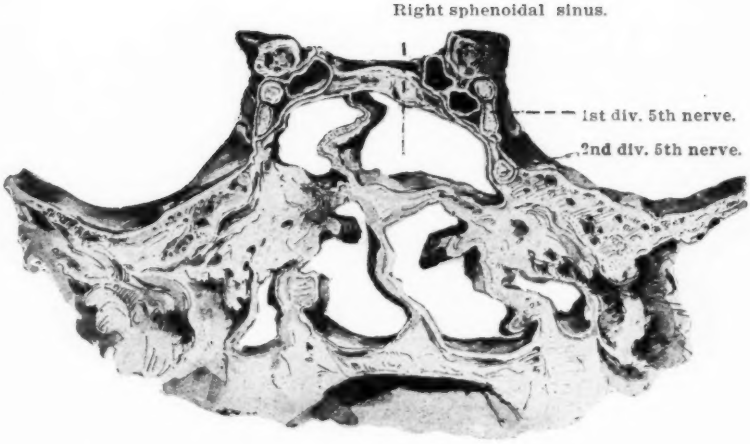


Fig. 8. (Two diameters.)

Cross section of sphenoidal sinus and anterior clinoid processes, showing first and second divisions of 5th nerve separated from sphenoidal sinus by thin bone.

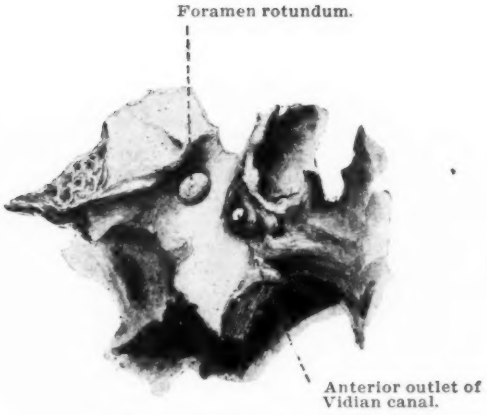
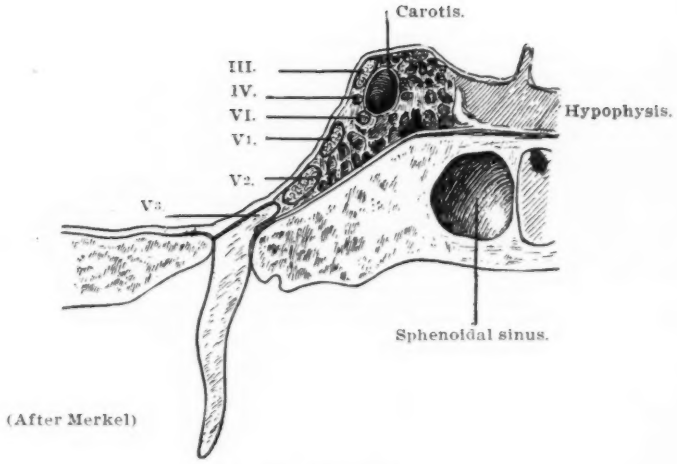


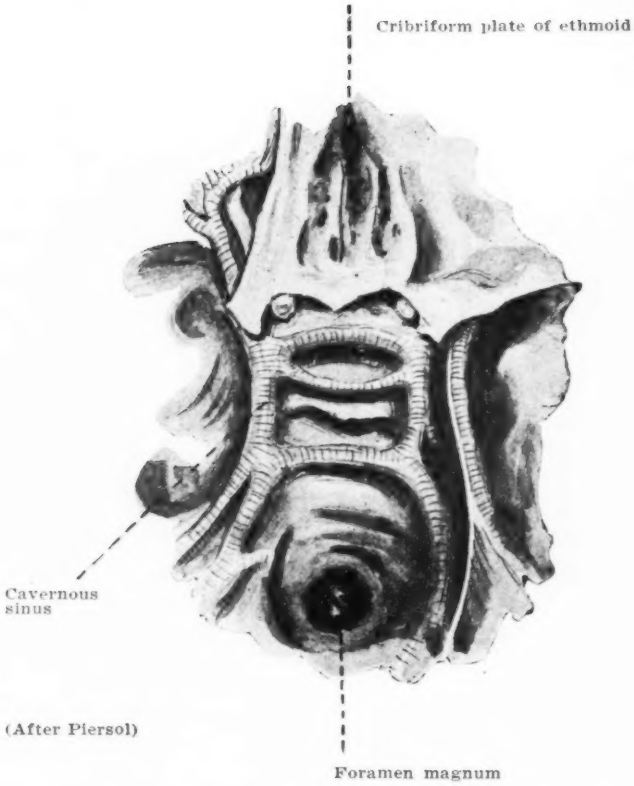
Fig. 9. (Two diameters.)

Anterior aspect of base of right pterygoid process, seen from in front.

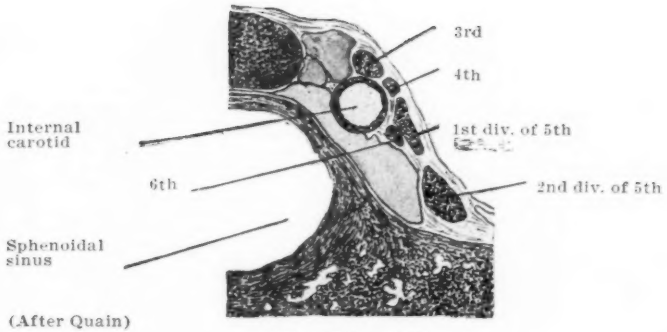


(Two diameters.)

Showing cavernous sinus and third, fourth and first, second and third divisions of the 5th and 6th nerves, hypophysis, sphenoidal sinus, and carotid artery.



Showing venous formation of cavernous sinus.



(After Quain)

(Two diameters.)

Showing cavernous sinus and third, fourth and first and second divisions of the 5th and 6th nerves, hypophysis, sphenoidal sinus, and carotid artery.

(After Rauber and Kopsch)

Showing venous formation of cavernous sinus, seen from above.

XLIX.

EXHIBITION OF OTITIC BRAIN ABSCESS CASES AND A MASTOID EXENTERATION BY NATURE.*.

By S. MacCuen Smith, M. D.,

Philadelphia.

Two of the cases that I have the pleasure of presenting to you today have been shown before the Section on Otology and Laryngology of the College of Physicians of Philadelphia, but are of sufficient interest, I feel, to be again brought to your notice. The first case, however, that of the eight-year-old child, is still under care and shows the results of an evacuated brain abscess formation, especially in the relief of the neurologic symptoms.

In the majority of my cases, I am impressed with the absence of diagnostic data that will enable us to state that an abscess formation is present, or, more particularly, its actual location, with these exceptions: The presence of word aphasia in a left-sided temporal abscess formation, and that indescribable clinical picture as seen in the facial expression. Indeed, I have been compelled in most instances to rely principally on these two symptoms, and regret that I am unable to definitely define the facial expression above referred to, but am sure you have all had a like experience.

Another symptom, which has been so constant in patients coming under my observation that I am inclined to believe it pathognomonic of an abscess formation, involving the temporosphenoidal lobe of the brain, is the very severe pain in the frontal region of the affected side. Indeed, this symptom has been so unfailingly present and so severe in the average case, that one might readily be deceived in thinking the abscess formation was in the frontal lobe.

---

*Read before the Eastern Section of the American Otological, Rhinological and Laryngological Society, at Philadelphia, February 22, 1913.

Of course, in the small minority of cases we are able, by certain tests, including the eye, blood and neurologic examinations, to state with more or less accuracy that a given patient is suffering from a brain abscess formation, and we might even be able to state its location with some degree of certainty; but if these same rules are applied to the average case, we would fall far short of proving our point. So, after all, personal observation, together with intuition, which comes only from large experience, must be depended upon to aid us principally in our diagnosis and localization of intracranial lesions complicating aural disease

I wish again to bring out one point which I feel is of the greatest importance: When possible, follow the path of infection through the tegmen antri or tegmen tympani, when searching for a brain abscess formation, as this not only favors drainage by gravitation, but also lessens the possibility of lighting up new infection by going through the skull at some other point.

Case 1.—D. P., aged eight years, was admitted to the Jefferson Hospital on October 24, 1912. Her family history was negative. Three years previous to this time the child had scarlet fever, complicated by an acute otitis media of the left ear, since which time the discharge has been constant except for two periods of about three months each, when it ceased. Just prior to the acute exacerbation the child would become especially irritable and mentally confused.

In the spring the child became restless and quarrelsome, lapsing from her naturally cheerful disposition to one irritable and morose, and growing progressively worse. About a month before she appeared for treatment, she fell and struck on her head, in the frontal region. Following this blow she had severe, persistent frontal and left temporal and parietal headache, with extreme nervous irritability, vomiting and intermittent delirium, principally nocturnal, and was able to retain but very little food. One week after the injury she had a convulsion.

Examination on Admission.—The child is fairly well nourished, temperature normal and pulse 53. Chest and abdominal examinations are negative, as are also the laboratory reports on blood and urine, and the neurologic and ophthalmologic reports.

Left Ear.—The canal is filled with a quantity of brownish-yellow, foul-smelling pus. There is moderate drooping of the superior and posterior wall of the canal, but no tenderness over the mastoid process. There is entire absence of the tympanic membrane and ossicles, and the mucosa lining the tympanic cavity is very red. The X-ray report, by Dr. Manges, shows the mastoid cells on the left side filled with pus. The right side is normal. Skull has very much the appearance of an internal hydrocephalus, but there is no evidence of brain abscess.

Operation.—A radical mastoid operation was performed on October 29th, or five days after admission. The cortex of the mastoid was very hard, and upon its removal the antrum was found to be almost obliterated. The sinus was in about a normal position. There was a carious exposure of the dura through the tegmen antri about one-fourth of an inch in diameter. The walls of the antrum and the few cells that still existed were covered with a peculiar membranous material.

The case followed an uneventful course of convalescence after the mastoid operation, but the marked irritability continued.

A Wassermann test was positive and the patient was placed on inunctions and potassium iodid, without any change in the clinical picture.

The child was allowed to return to her home in central Pennsylvania in about one week, owing to her extreme homesickness, but on the following day she had a second convulsion, and was immediately brought back to the Jefferson Hospital, where she was kept under observation for two weeks, when she developed measles and was sent to the Municipal Hospital, whence she returned to her home one month after the mastoid operation.

For a fortnight her condition was much improved, when suddenly, without apparent cause, the severe headache over the left frontal region returned and her mental state became noticeably worse. She could not recall the names of people whom she had known all her life, nor associate names with common objects. For instance, she called a knife a ring, but said it was used to cut. Her chief complaint was headache.

The patient was returned to Philadelphia and admitted to the Germantown Hospital six weeks after the mastoid opera-

tion. An examination showed the ear to be in good condition. The temperature was 98.8°, the pulse 98 and respiration 26. The urine and blood examinations were negative.

The eye report showed pupils equal, reaction normal except outward rotation of the right eye, which was practically nil. Eye grounds, media clear; discs swollen and margin everywhere obscured; best seen with a + 5. Retinæ edematous, blurring the blood vessels. Small veins tortuous. No hemorrhages seen. (Choked disc.)

The neurologic report showed no exaggeration of patellar reflexes. No ankle clonus. Babinski slight on both sides. Romberg's sign present on both sides, worse on right. Some rigidity of muscles at nape of neck. Masseter muscles contract firmly on both sides. There seemed to be no paralysis involving facial nerve on either side. Paralysis of abducens on each side. Grips equal, but not vigorous on either side. Patient was extremely irritable and suffered from almost constant frontal headache.

Four days after admission to the hospital, under general anesthesia, we reopened the mastoid wound, with a view of exploring the interior of the skull, hoping to locate and evacuate the suspected brain abscess. The process of repair was found to have gone on very rapidly. The exposure of the dura was enlarged by the removal of a lateral plate of bone for a distance of about one-half inch. There was no pulsation discernible in the dura and it seemed to be under considerable pressure. The dura was incised and a pair of Jackson forceps was introduced towards the temporosphenoidal lobe, along the avenue of invasion, for a distance of about one and one-half inches, when there was a free escape of a greenish, odorless pus, two ounces of which were collected in test tubes and it was estimated that about another ounce escaped. A drain was introduced, and the patient returned to her bed in a decidedly exhausted condition, it being necessary to employ normal salt solution intravenously, after which she reacted well. An examination of the pus showed few organisms present, staphylococcus pyogenes albus predominating. No tubercle bacilli.

Twelve days after the operation the patient was totally relieved of all suffering, her mental state was virtually normal,

and she could recall many incidents that occurred several years previously.

Six weeks after the brain abscess operation, notwithstanding our efforts to keep the external wound open, it had closed sufficiently to require reopening, which was done under general anesthesia.

Case 2.—W. S., aged thirty-seven years. When about twelve years of age he had an attack of scarlet fever, complicated by a suppurative otitis media of the left ear, which had been recurrent ever since, or for about twenty-five years. The longest period between attacks was from two to three years. During this time he maintained a state of good health. On two different occasions, about ten years ago, a polypoid mass was removed from the ear.

Examination showed the left ear well filled with a brownish-yellow, foul-smelling discharge, escaping through a large perforation in the posterosuperior quadrant of the drumhead, involving almost one-half of this part of the membrane. A large mass of granulation tissue practically filled the tympanic cavity. Considerable discharge was also escaping through a large carious opening about the middle of the posterior osseous canal, through which a probe was passed directly into the antrum, revealing much carious bone. The patient suffered from attacks of vertigo about once in each week, of sufficient severity to cause him to fall unless he promptly obtained some support,

The patient believed that he was entirely deaf in this ear, although he could hear the tuning-fork in close proximity to the concha. Bone conduction was greatly reduced from normal. No tenderness was elicited on pressure over the mastoid.

Because of his apparent good health and the absence of suspicion of intracranial lesions, the usual preliminary examinations of blood and eye grounds were not made.

Operation, March 4, 1909.—A radical mastoid operation was performed at the Jefferson Hospital. The cortex was found to be very thick and hard, with the exception of the mastoid tip, which was soft and necrotic. The antrum was reached with difficulty, on account of the dense, eburnated bone. As soon as the antrum was opened a quantity of pus gushed out, as though it had been under some pressure. When this was cleaned away the pus was seen to escape through a carious

opening in the tegmen antri and through the dura. A probe was introduced through this opening into the interior of the skull, the sinus apparently leading into the temporal lobe. A pair of Jackson forceps was then introduced and separated, permitting the escape of additional pus. Proper drainage was provided and the patient made a slow but uninterrupted recovery.

The chief point of interest in this case is that the patient apparently suffered no inconvenience at any time from this pus formation in the interior of the skull, unless we may attribute his spells of vertigo to this cause. It would, of course, have been most interesting to have had a record of his visual fields and a differential blood count.

The ear at this time, four years after the operation, looks perfectly well in every respect. Complete dermatization took place within one year, and the patient now hears the tuning fork at about two and one-half inches.

Case 3.—E. C. B., aged twenty-five years. Car inspector. Patient had measles when two or three years of age, and complicating this disease an acute suppurative otitis media of the left ear. The discharge in this ear continued recurrently up to one year ago, when the patient applied for treatment at the Jefferson Hospital, and just at that time was especially active and profuse. For about a month he had suffered from vertigo, which was increased by bending the body or by quickly turning the head, in any direction.

When first examined the external auditory canal was well filled with a collection of pus and desquamated epithelium. Upon the removal of this mass we found an entire absence of the membrana tympani and ossicles, they having become disintegrated and escaping in the discharge probably some years ago. A bent probe introduced into the antrum shows that the cells comprising the tip have become obliterated through sclerosis. The greater part of the superior and posterior osseous canal is destroyed, from the tympanic cavity back to the mastoid antrum, all of which, except the tympanic end of the eustachian tube, seems thoroughly dry and shows complete dermatization.

Bone conduction is little, if any, reduced. Hearing for C tuning fork, aerial conduction, is only one-half inch.

Various tests indicate that the patient has a marked vesti-

bular disturbance, more especially involving the superior and horizontal semicircular canals. The whole picture shows in an unusual manner nature's efforts to provide for evacuation of the inflammatory débris by performing essentially a radical mastoid operation.

REPORTS.

X-Ray.—Left mastoid is of a chronic sclerotic type, with considerable bone defect in the region of the antrum cells. The lateral sinus is not superficial and does not appear to be far forward. The mastoid process on the right side is apparently normal. (Dr. Manges).

Neurologic.—We find no neurologic symptoms in this patient other than deafness and vertigo. There are no central symptoms. He is not hysteric nor neurasthenic. (Dr. Price.)

Ophthalmologic.—Patient is suffering from compound myopia and requires reglassing. Otherwise the eyes are healthy. (Dr. Le Fevre.)

L.

A REPORT OF THREE UNUSUAL CASES OF STAMMERING.*

By G. Hudson Makuen, M. D.,

Philadelphia.

Stammering is like many similar nervous affections or diseases in that it may be divided into three more or less distinct stages: the prodromal, the acute, and the chronic. The prodromal stage is found in early childhood, and it is characterized by rapid and blustering speech. The acute stage comes in later childhood, and it is characterized by a marked hesitation in speech, which is usually unconscious, although accompanied by varying degrees of spasmodic muscular contractions and contortions. The chronic stage follows hard upon the other two as the natural result of a continuance of the affection, and it has all the symptoms of the other two in a somewhat aggravated form, together with certain important mental and emotional complications which always accompany a full, conscious realization of the condition.

The prodromal stage is one from which many children pass without serious difficulty, especially when they have some intelligent help; the acute stage is the forerunner of more serious trouble, and the chronic stage is the full development of the affection with all its varied complications.

Every case of stammering is unusual because no two are exactly alike, but some cases are more unusual than others. The three which I am about to report are alike in that they are all in the chronic stage, two of the men afflicted being forty years of age and having stammered for more than thirty-five years, and one of them being thirty-two years of age and having stammered ever since he can remember.

Case 1.—E. K., a physician, forty years of age, has stam-

*Read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, February 22, 1913.

mered for thirty-five years. His stammering varies in degree and it has increased in intensity. There is no history of hereditary influences, nor was the patient associated as a child with any similarly afflicted. He had whooping cough at an early age, but it was not sufficiently severe to be a factor in his condition.

The patient is in good health and possessed of all his faculties. He is nervous and excitable, although he claims that this condition is a result rather than a cause of his stammering. He is of average mentality, has a good memory, is strongly initiative, and does not appear to be lacking in attention, although his powers of concentration and volition are unstable. His family history reveals nothing of a positive character except that his mother died of tuberculosis. The patient himself is unable to recall any particular incident in his life that may suggest a cause for the beginning of his speech difficulties. There is no peculiarity about his writing. He stammers somewhat when he attempts to read aloud, he stammers very much in conversation, and he is quite unable to speak at all in public. The defect does not appear in singing.

He claims to have had no subjective nose, throat or ear trouble, although the septum is markedly deflected toward the left nostril, causing considerable intranasal pressure and pharyngeal irritability; a condition, by the way, which I have found to be very common with stammerers. More than 90 per cent of my cases have intranasal pressure with irritability of the pharynx.

The patient had taken a course of treatment in Philadelphia and had been twice to a Detroit school for stammerers, but he had received no real benefit whatsoever. His conversational speech, when he was able to say any words at all, was drawling and painful to listen to, and it was accompanied by various motions of the hand and forearm, such as beating time and drawing figures of eight, which maneuvers he claimed made it possible at times for him to surmount his difficulties to some extent, and occasionally at least to converse with his friends because they enabled him to forget his stammering and thus to banish the fear of it.

His own experience had led him to the belief that stammering is a "phobia," pure and simple, and that there is no

help for the affection except through the removal of fear. He was convinced also that all training of the organs of speech must be worse than useless, because it tends to fix the attention upon the difficulties and thus to increase the fear. He was sure that he could speak well if he could banish fear, but he was equally sure that there was no power on earth that could help him to do it.

He was in Philadelphia about two weeks, during which time I saw him occasionally and tried to convince him of the error of his ways and the falsity of his theories, but I failed utterly because he was so disinclined to do the amount of work which would have been necessary in his case to enable him to acquire volitional control of his organs of speech, and thus prove to himself that there was no basis for his fear. It was of no avail that I quoted Emerson where he says, "The antidote to fear is knowledge." The fear of stammering always vanishes when the patient has acquired a good working knowledge of the mechanisms of speech and a voluntary control of them through systematic and persistent practice.

This man, however, was in the unfortunate condition of mind, which is by no means uncommon in these cases, of being unable to make any persistent effort to do what seemed to him a great amount of work, because of his doubt or lack of confidence in the results to be acquired. And who can blame him? He had had three unfortunate experiences with other methods for a cure. He had given his money, his time, and a certain amount of energy to the work without receiving an adequate return, and his confidence was not only shaken but absolutely shattered. I could not persuade him to try again. It was in vain that I pointed out to him the difference between the methods which he had used and the one which I was advocating. I tried to show him that the former were merely tricks to divert his mind from the difficulties and thus enable him to overcome them, while the latter system, or the one which I would have him adopt, was along physiologic lines and consisted merely in an effort to supplant his defective speech with normal speech, built up on scientific principles. I was utterly unable to convince him that the end would justify the means, and he went away somewhat in the condition of the man who said that "it was happier to be miserable than to be happy without being miserable."

Case 2.—Mr. R., also forty years of age, has stammered for thirty-six years. His stammering is not increasing because, as he said, it cannot be worse. It was of gradual development and it began spontaneously. When a child, he says, he was "saturated with the Bible," and he was once greatly disturbed by overhearing a conversation in which it was predicted that the world was soon coming to an end. He gives no history of relatives having stammered in their speech, but he says that his mother, who was also sensitive on the end of the world question, had something akin to stammering in her fingers while playing the piano. He has one younger sister, who has no defective speech whatsoever. He had whooping cough and measles during infancy, and he suffered from some intestinal colic when very young. His memory is fairly good, but his powers of concentration are poor.

The patient's father developed epileptiform attacks late in life, which interfered with his speaking and writing during the last two years of his life.

The patient is exceedingly nervous and excitable, a condition which he thinks he inherits and which is largely responsible for his trouble. In 1884 he studied some months with a negro who had himself been cured, and thus he got some temporary relief. In 1890 he was in a school here in Philadelphia for the cure of stammering, and again in 1894. In November of 1911, he consulted Dr. Coriat of Boston, who attempted some mental or psychic method of treatment, but he was unable to put him in the hypnotic state, or to find any subconscious cause for his condition. The theory is now being advanced by certain neurologists or psychologists that stammering is due to a "forgotten reminiscence," such, for instance, as this man's fright of early childhood with reference to the ending of the world, and that this forgotten reminiscence must be recalled or brought within the domain of the consciousness before the stammerer can be permanently cured. Professor Munsterburg of Harvard was nearer the truth in the matter, however, when he said he had long since ceased to experiment with stammering, because the psychic method halts when it reaches the physical obstruction.

Case 3.—G. G., aged thirty-two years, employed in a wholesale drug house, had stammered ever since he could remember. His father was an alcoholic and died in an insane asylum.

When he was a baby, his father came home intoxicated one evening and upset the cradle, greatly frightening the child and every one in the house.

The patient had none of the infectious diseases of childhood, and there was no one else in the family who had been similarly afflicted. He was mentally alert and fairly bright, but inclined to be introspective, moody, and at times even morose. He seemed to brood over his condition, but not, as I thought, excessively or more than he could bear, and he began his treatment with us in a fairly satisfactory manner, making very good progress, indeed. His sister called upon us after two weeks had elapsed and expressed herself as greatly pleased. She said that he talked better and he was much less nervous. After about five weeks, however, he made a little visit home, and his uncle showed some disappointment and displeasure, because as he said, he seemed not to be entirely cured.

This greatly discouraged the patient, and he came back to us in a very nervous and excited condition. My assistant, at whose home he was staying, said that she was anxious about him because he seemed at times not to be able to control himself or to do his work satisfactorily. He would draw his hand across his forehead and stare into vacancy in a somewhat excited manner, while all the time trying to do the things which were required of him. I myself had several interviews with him in which he seemed to be entirely rational, although it was with difficulty that I was able even temporarily to help him out of his condition of despondency. I made no attempt to hypnotize him, but I used every other psychic measure at my disposal to bring about a more cheerful and hopeful attitude with reference to his work. Just as I thought we were succeeding in our efforts, he suddenly, and without giving warning or notice either to me or to his hostess, packed his bag and went home. He immediately wrote me to the effect that he had decided to give up the treatment for a while at least, to which I replied that perhaps he would feel like returning later; but in about a week, I received a letter from his mother stating that her son had been greatly disturbed because he had not been able to more speedily overcome his speech difficulties, and although she tried in every way to cheer him up, he absolutely refused to be comforted, and after

eating his breakfast one morning, he left his home, presumably to go to his place of business, but he went instead to a neighboring park and took his life with a revolver.

I mention this fact to show the awful mental suffering that sometimes attends this affection. I have had several patients tell me that self-destruction seemed to them at times to be the only real way out of their difficulties. This feeling is always increased with each failure to overcome the stammering, and in as much as about 90 per cent of those who attend the various institutions for the cure of the affection fail in reaching a solution of the problem of good speech, it is a question whether the schools for stammering on the whole may not be doing more harm than good.

Moreover, there is a common belief—and it seems to exist even in the medical profession—that children grow out of stammering, but this I think is a thing that never happens. It is true that the habit of stammering is often somewhat overcome in later life, but such improvement in speech is almost always the result of assistance from some source and great painstaking effort on the part of the individual. Children do not of themselves grow out of the affection, but they grow into it, and its awful consequences are well illustrated by the three cases I have described.

LI.

DIAGNOSIS AND TREATMENT OF DIPHTHERIA.*

By Joseph B. Greene, M. A., M. D.,

Asheville.

The fact that diphtheria has received such scant consideration from the laryngologists of the country is my reason for bringing this subject to the attention of this society. One cause of this apparent neglect is the fact that in many of our large cities patients with this disease are promptly transferred to contagious hospitals. This procedure develops a certain number of physicians in the hospital highly skilled in diagnosticating and treating diphtheria, and who are especially skilled in performing intubation and tracheotomy, which measures are at times so necessary for the saving of life. In consequence of this prompt transfer of patients, laryngologists of the cities see fewer cases, and are, as a result, called upon less frequently than formerly to perform intubation or tracheotomy. The early use of antitoxin has likewise materially reduced the number of cases requiring operation.

In our smaller communities the general practitioner still, to a large extent, attends his diphtheria cases alone, though I know of no disease where the internist and laryngologist can, to a better advantage to the patient, work hand in hand. The throat man can often be of service in determining the extent of the lesion, particularly as to the nose and larynx, and likewise diagnosticating early complications, as a beginning otitis media. The laryngologist, on account of his being accustomed to examine and treat the larynx, is better prepared to perform intubation when needed.

The diphtheria organism may attack any mucous membrane, and likewise the abraded skin, as in eczema and wounds. Diphtheria of the pharynx is the most common form, and the

*Presented as candidate's thesis at the nineteenth annual meeting of the American Laryngological, Rhinological and Otological Society, held at Washington, D. C., May 8-10, 1913.

one to which I shall first direct your attention. Unfortunately, there is no method of diagnosticating, with positiveness, diphtheria from "ordinary sore throat" socalled, except by finding the characteristic organism, Klebs-Loeffler bacillus. Even when a culture is taken, one negative finding does not exclude diphtheria, if the clinical symptoms are suggestive, and in the absence of a positive finding, the patient should not be denied the benefit of antitoxin. The case which is reported later illustrates this point. The cause of a negative finding in cases of true diphtheria is not always the fault of the laboratory. It may be due to a failure to touch accurately the edge of the membrane, perhaps due to an imperfect view of the throat. One has often to contend against a struggling child, and frequently, too, in a poorly lighted room. It is also necessary to bring the inoculated part of the swab in contact with the surface of the blood serum. The use of antiseptics within an hour or so of the time of taking the culture destroys all chance of a growth on the serum tube.

The diagnosis of diphtheria has been relatively easy since Klebs, in 1883, discovered the organism in the diphtheritic membrane, and Loeffler in 1884, one year later, isolated the organism—hence Klebs-Loeffler bacillus. It is a small rod of irregular size and shape. It stains unevenly with Loeffler's alkaline methylene blue, giving at times a beaded appearance. This staining peculiarity is of great diagnostic importance, and is more marked in a culture twelve or more hours old than in a young culture, or from a swab of the throat. There is a tendency for the organism to assume a parallel arrangement, which has served me well in doubtful cases. This is more noticeable from a culture than from a swab. The organism is stained by Gram's method. As a differential stain, there is probably none better than Neisser's. Here the peculiar granules are stained dark blue, while the body of the organism assumes a light brown color. The most suitable culture medium is blood serum. On this it grows rapidly at body temperature, distancing the common pus organism, so, at the end of six or eight hours, a diagnosis can usually be made with the microscope. In some cases a staining of the swab will give valuable aid in the diagnosis, though a culture will be found more reliable.

Although the bacteriologic findings are of first importance

in making a correct diagnosis, as before stated, when in doubt give antitoxin, if the case is at all severe. In primary laryngeal cases a negative finding is of little value if the swab is only taken from the pharynx. A laryngeal applicator could in these cases be used to advantage.

Cultures should be taken of all sore throats, particularly in infants and children. It is impossible to state otherwise, with accuracy, whether the case be one of simple tonsillitis or diphtheria. A correct diagnosis is important, not only for the safety of the child, but also from a public health standpoint. The following case will serve to illustrate two points—in the first place, the importance of not waiting for a positive finding before giving anitoxin, and secondly, the necessity of administering large doses of antitoxin.

Patient, L. C., age seven years. First seen by me at Asheville, N. C., in consultation with Dr. Powell, on Sunday night, July 2nd, the fifth day of her illness. The attack began with all the clinical appearances of simple tonsillitis. The culture negative. On Saturday, the day prior to my visit, the temperature was normal till 6:30 p. m., when it registered 99.5°. When I first saw her on Sunday night the picture had changed, for the membrane had spread from the right tonsil to the anterior pillar of the same side. A patch was also seen on the left tonsil. Temperature 103.5°. Neck very much swollen, and head extended to facilitate breathing. Patient showed signs of severe toxemia. Administered at once 10,000 units of antitoxin. The following morning patient no better; given 20,000 units of antitoxin. That afternoon, Monday, patient still dangerously ill. Culture positive for the first time. Seen with Dr. Dunn, and 40,000 units given. The next morning, Tuesday, seen with Dr. Briggs, patient slightly better, but still dangerously ill. Repeated 40,000 units. That afternoon patient much better, and only 20,000 units given. Small doses of antitoxin were continued for a few days, till all signs of membrane had disappeared. She was given, during her illness, 170,000 units of antitoxin. Her recovery was complete and uneventful, except for some paralysis of her palate and eye muscles. These had all disappeared at the end of nine weeks. Her pulse continued rapid for some time, which required absolute quiet. Stimulants were given as indicated.

Hoffman's bacillus closely resembles the diphtheria bacillus,

though it is somewhat shorter and thicker. It does not show the polar bodies by Neisser, nor is it pathogenic to guinea pigs.

Bacillus xerosis,[1] as recently described, cannot be distinguished from Klebs-Loeffler bacillus. It is further said that bacillus xerosis forms acid from saccharose, not from dextrin; while bacillus diphtheria forms acid from dextrin, and not from saccharose. However, the final test in differentiating bacillus diphtheria from all socalled "pseudodiphtheria" bacilli is by animal inoculation. This slow method of differentiation can only be of use from a public health standpoint.

The clinical diagnosis of diphtheria is often difficult. It is particularly so in mild cases affecting the tonsils alone, and also where the larynx is primarily affected. Temperature is of little value in making a diagnosis of diphtheria. It is frequently not high, ranging from 99 to 100° F. Strange as it may seem, the temperature may be normal in cases of severe toxemia. A sudden rise in temperature suggests a beginning otitis media or pneumonia. The pulse, like the temperature, is a poor guide in the diagnosis, for it may vary from a slow pulse to a very rapid one. The extremes, as a rule, are of serious prognostic significance. The volume of the pulse soon becomes small, owing to a myocarditis. The depression is great in cases of severe toxemia, though in mild cases this need not be the case. It is too often said by the attending physician: "The patient is not sick enough for diphtheria." Follicular tonsillitis usually makes one seem sicker than true diphtheria. The temperature is usually higher. Before the appearance of the membrane there may be observed a peculiar plum color of the mucous membrane of the pharynx, particularly evident on the soft palate. There may be considerable edema present. Valuable time in the treatment may be saved by this early diagnosis. The membrane is at first of a grayish white color, though not unlike the membrane due to the pus organism. The cases of septic sore throat which occurred in Boston[2] from the milk epidemic of 1911 had some clinical resemblance to diphtheria. The bacteriology was different. The situation of the membrane is of great diagnostic importance. If found on the pillars, or any part of the pharynx other than the faucial tonsils, our suspicions should at once be aroused that we are not dealing with a case of simple sore throat. The odor in septic diphtheria may be foul. There

is usually seen a peculiar red margin at the edge of the membrane, which has served me well for diagnosis in certain cases. The use of violence in removing the membrane to see if bleeding takes place is of little value, and is unjustfiable. Mucus in the throat may be gently swabbed away without harm to the patient. Albumin in the urine is suggestive of diphtheria. Early glandular involvement is a suspicious symptom of diphtheria, though the Boston milk epidemic of 1911 showed this symptom very frequently. There may be edema of the neck, which may extend down over the chest. This is strong evidence of diphtheria, and indicates a severe type of the disease. These are cases requiring large dosage of antitoxin.

Nasal diphtheria is the most contagious form, and from a public health standpoint is of the first importance. The membrane may be in the nose proper, or the nasopharynx. In the latter situation, that is, in the nasopharynx, the symptoms are more severe, owing to a rapid absorption through a bountiful lymph drainage. On account of the difficulty of inspecting the nasopharynx of very young children, the diagnosis of this type of diphtheria must usually be made from the symptoms and finding the characteristic organism. There is noted a serous or serosanguinous discharge from the nose, which may excoriate the upper lip. When the membrane is in the nose one can usually see it upon rhinoscopic examination. Frequently the membrane may be confined to one side of the nose, and then you get a one-sided discharge which simulates a foreign body in the nose. There may be nasal hemorrhage of some severity. The patient, if old enough, complains of a stopped-up feeling of the nose. The symptoms may persist for a long time, and may be so mild as not to be suspected as being diphtheria. As before stated, these cases are very contagious and become a menace to public health.

From autopsies performed by Councilman and Mallory at the South Department, Boston, it is probable that the sinuses of the nose, particularly the antrum and sphenoid, harbor the diphtheria organism for a long time. There may be an extension of the membrane from the nose to the ear, pharynx, larynx, or eye.

Diphtheria of the eye is a serious condition, particularly as to sight, and frequently results from an extension through

the tear duct, though it may be due to accidental inoculation from other regions.

When the larynx is primarily involved it is difficult of diagnosis, especially in very young children. On account of the difficulty of inspecting the larynx in children by means of the indirect method, the laryngeal specula, as devised by Mosher and Jackson, are useful for direct laryngoscopy. The symptoms of laryngeal obstruction from a diphtheritic process come on slowly, and in this respect differ from a foreign body, and also spasmodic croup. At first there is hoarseness, followed by aphonia, evidenced in infants on attempting to cry. In a few hours this obstruction becomes greater, and we get signs of the patient breathing through a stenosed larynx, such as stridor, both inspiratory and expiratory in character, rigidity of the sternocleidomastoid muscle, and retraction of the abdominal muscles and supraclavicular spaces. There may be pallor instead of cyanosis, a fact which is easily overlooked when the question of giving relief by operative interference is considered. When the obstruction is lower down, as a foreign body in the bronchus or bronchial glands, there is more expiratory stridor. The auscultory signs of the chest in cases of foreign body are the absence of normal respiratory sounds over the area where the entrance of air is obstructed. When the obstruction is high up, as in retropharyngeal abscess, the stridor is more inspiratory in character. The diagnosis of retropharnygeal abscess can best be made by palpation with the index finger.

Catarrhal spasm of the larynx, or spasmodic croup, as it is sometimes called, may be mistaken for laryngeal diphtheria, but the former usually comes on suddenly at night, and there may be a history of previous attacks. The temperature is usually lower in spasmodic croup, though too much reliance must not be placed on this point in the diagnosis. Improvement usually follows in a few hours after appropriate treatment, not the case in laryngeal diphtheria unless relieved by operation.

Acute laryngitis in infants simulates diphtheria very closely. The constitutional symptoms are usually milder, except the temperature, which may be higher than in laryngeal diphtheria. Aphonia is not so marked. If there be a patch on the pharynx, glandular enlargement of the neck, or albumin in the urine,

think of diphtheria. Direct laryngoscopy should be practiced in all doubtful cases.

Pneumonia resembles laryngeal diphtheria in the marked dyspnea, but there is usually no stridor. There can be elicited the physical signs of pneumonia, and probably a leucocytosis.

Jackson[3] has described a laryngotracheitis due to the influenza bacillus, which can be differentiated from diphtheria only by direct inspection. There is no membrane or Klebs-Loeffler bacilli present.

In distinguishing the laryngeal symptoms of measles, we should look for Koplik's spots, which appear before the skin rash. The eye and nose symptoms appear early in measles. A culture from the throat should, of course, be taken.

Diphtheria may be a complication of measles. In fact, it complicates this disease more frequently than any other.[4] Early laryngeal symptoms point to measles alone, while a later stenosis of the larynx in measles suggests diphtheria as a complication.

The diagnosis of diphtheria from scarlet fever, when a membrane is present, can only be made by the microscope. The general redness of the fauces, the appearance of the tongue, and perhaps the skin rash in scarlet fever may assist in the clinical diagnosis. One should be on the lookout for a complication of scarlet fever with diphtheria.

Vincent's angina has been very clearly and fully described by Place.[5] It may be mistaken for diphtheria. It is more chronic in nature, and usually shows a dirty patch on the tonsil or elsewhere in the mouth. Place says: "It differs from diphtheria in being always an ulceromembranous process, which diphtheria never is primarily." The finding of the bacillus fusiformis and the spirillum gracillis confirms the clinical diagnosis. Syphilis of the nose and pharynx may simulate diphtheria, but with the finding of the specific spirochete and the Wassermann reaction the diagnostic difficulties should be less.

Inherited syphilis of the nose in a young child may be mistaken for diphtheria, but there are usually other evidences of syphilis. It is less acute in character and there is the absence of Klebs-Loeffler bacilli.

Secondary syphilis of the throat is characterized by a thin layer of necrotic epithelia with a well defined border. There is

usually less constitutional involvement, though there may be fever with syphilis.

Later forms of syphilis of the throat are characterized by a deep ulcer with a very much inflamed border. The appearance is out of all proportion to the symptoms, for there is usually little pain on swallowing. With the Wassermann reaction in syphilis and the finding of Klebs-Loeffler bacilli in diphtheria, there should not long exist doubt in the diagnosis of syphilis and diphtheria.

Traumatic exudates from operative procedures, as tonsillectomy, simulate diphtheria. A close inspection and a history of the case should remove all doubt. It should be remembered, however, that Klebs-Loeffler bacilli may be a complication of an operative procedure, so if there exist constitutional symptoms out of proportion to the appearance of the wound, a culture should be taken.

Leptothrix mycosis may be distinguished from diphtheria by its chronic course, often without symptoms. The projections on the tonsil are conical and show a growth of the leptothrix fungus.

In all lesions simulating diphtheria the microscope should be promptly used in clearing the diagnosis.

PREVENTIVE TREATMENT.

Patients should, if possible, be sent to an infectious hospital. In lieu of this, isolation and quarantine should be rigidly enforced at home. Immunizing doses of antitoxin, 500 to 2,000 units, depending somewhat on the age, should be administered to all exposed children. Adults should be carefully watched, as to their noses and throats.

There is hardly a question but that epidemics of diphtheria are kept up by "carriers," so, as a preventive measure, these cases should be sought for and isolated. Seligmann[6] lays stress on this point, and also speaks of nasal diphtheria being liable to convey the infection for a long period of time. Diseased tonsils and adenoids are especially liable to harbor the organism. The writer,[7] in 1908, removed diseased tonsils and adenoids for the purpose of getting rid of the diphtheria organism in a "carrier," and soon the patient was rid of the infection. Since that time this method has been frequently practiced in certain localities with marked success. It would

not be advisable to practice this method as a routine procedure, but is suitable for certain selected cases. An immunizing dose of antitoxin should be given at the time of the tonsil operation, to prevent infection of the wound with Klebs-Loeffler bacilli.. Scheotz, a Danish physician, some years ago, recommended spraying the throat with a living culture of staphylococcus pyogenese aureus, to rid the throat of diphtheria infection. Page,[8] Lorenz and Ravenel[9] have practiced the overriding of diphtheria throats with staphylococcus pyogenes aureus, and report good results. As the two organisms are found growing side by side in throats, and can likewise be grown together in culture tubes, it is not easy to understand the reason for the disappearance of the diphtheria organism by this method of treatment. It would seem as if there is an element of danger in a spray of living staphylococci. Inasmuch as "carriers" have, as a rule, abnormal throats or noses, it would seem more rational to treat these abnormalities with a hope of ridding the patient of diphtheria bacilli. Place tells of a "carrier" who got rid of his infection when a foreign body was removed from the nose. At least two negative cultures, and probably three, should be required before release from isolation. The writer saw last year, while working at the South Department, Boston, a child who had been in the institution more than a year, awaiting the required negative cultures. Diphtheria vaccines[10] have been used with some success in ridding "carriers" of diphtheria bacilli.

SERUM THERAPY.

Under treatment, I shall consider first, antitoxin, for it is by far the most important. It is an absolute specific when given in time and in sufficient dosage. Of the 241 physicians, nurses, and attendants contracting diphtheria at the South Department, Boston, not one succumbed. It is not necessary to consume your time in offering proof of the efficacy of antitoxin in this formerly much dreaded disease. I assume that every member of this society is a believer in the life-saving value of antitoxin. It should be given early, and in sufficient dosage to produce the desired effect. The size of the initial dose should vary from 5,000 to 50,000 antitoxin units, and more in the malignant cases which have existed for some time. As McCollom rightly says, the age, over two years, has noth-

ing to do with the size of the dosage. It is a chemical reaction, and not a physiologic one, so there is no reason to doubt but that a young child may have as many toxin units as a fully grown man. They certainly have a lessened resistance on account of their tender years. The antitoxin is, in no sense, an antiseptic, but in neutralizing the toxin it gives the leucocytes and other protective forces a chance to get in their work.

Textbooks have been very inadequate in the size of the dosage recommended. Tyson, in his "Practice of Medicine" recommends 1,000 to 2,000 units, depending upon the severity of the attack. Ballenger recommends 2,000 to 3,000 units in what he terms ordinary diphtheria; 3,000 to 5,000 units in laryngeal cases. McCollom, in his classical paper on diphtheria in Osler's Modern Medicine, says: "If there is a very extensive membrane when the patient is seen for the first time, 8,000 to 10,000 units should be given, repeated every four or six hours, until the characteristic effect of the serum is produced, namely, the shriveling of the membrane, diminution of the nasal discharge, a correction of the fetid odor, and general improvement."

Since the above was written, the dosage at the South Department, Boston, has been increased, and the mortality likewise reduced. It may be of interest to state that the mortality at this large infectious hospital, from 1888 to 1894, the year of the introduction of antitoxin, was 43.2 per cent. From 1895 to 1904 inclusive, it was 11.48 per cent. For the year 1912 it was 7.6 per cent, including laryngeal and moribund cases. The greatest reduction was in the very severe cases where massive doses of antitoxin were administered.

Both nasal and laryngeal diphtheria require large doses of antitoxin. Abraham Levinson[11] says that the curative dose in nasal and laryngeal cases should not be less than 25,000 units. Diphtheria of the eye, on account of the danger to sight, requires large doses of antitoxin. It is far safer to give larger doses than is required to neutralize the toxin, which is so destructive to the vital cells, than to leave a portion free to exert its deadly effect on the tissues of the body. There is no way of estimating, with accuracy, the amount of toxin requiring neutralization, but the amount depends somewhat upon the duration of the illness, the extent of the surface involved, and, lastly, the virulence of the organism. A mild case of

pharyngeal diphtheria should receive, as a single dose, from 5,000 to 10,000 antitoxin units; a moderately severe case of several days' standing should receive 20,000 to 25,000 units, and a severe case, 50,000, and more if the case seems desperate. The size of the dose can soon be reduced and smaller doses continued until the membrane has completely disappeared. Place strongly advises getting all the required antitoxin in during the first three days of the treatment, even if 500,000 units are required. This dosage, of course, refers only to the desperately ill cases.

Ehrlich says two poisons are produced by the growth and multiplication of the diphtheria bacillus. To one he gives the name of toxin, and to the other toxon. To the former he attributes the early acute symptoms, and to the latter, the toxon element, he attributes the late paralyses. The affinity is stronger between the toxin and antitoxin than between the toxon and the antitoxin, hence the importance of having a surplus of antitoxin to neutralize the toxon element after the toxin has been chemically disposed of.

There is what is termed a precocious form of palatal palsy occurring about the fifth day of the disease. This is not so serious as the late forms, occurring usually within the first three weeks of the illness, but the early form is somewhat an index to the severity of the illness.

Rosenau and Anderson[12] have shown by animal experimentation the uselessness of giving antitoxin with a hope of influencing the paralyses occurring late in the disease.

My favorite situation for injection of the antitoxin is in the loose areolar tissue of the back, near the angle of the scapula. This region is relatively free from the sensory nerves, is protected from vital parts, and lastly, the operation is not witnessed by the patient. This last point is quite important from a psychic standpoint. The site selected should be sterilized, and the situation should be changed from time to time, as there is usually a local reaction.

Fritz Meyer[13] strongly recommends intravenous injection of antitoxin in serious cases—says much time is saved in the absorption. Eckert[14] and Hoesch[15] and others advise intramuscular injection as being more rapid of absorption than the subcutaneous method.

DANGER OF ANTITOXIN.

The danger of antitoxin has been grossly exaggerated. That a few deaths have taken place following its administration cannot be denied, but they are too infrequent to be seriously considered in view of the dangers of diphtheria when left untreated by antitoxin.

Park[16] says that "In over 100,000 patients immunized (New York) since the introduction of antitoxin, there has been but one known fatality due to the serum injected. This child suffered from status lymphaticus and died shortly after an injection of 1000 units." Place says that more than 100,000 doses of antitoxin have been administered to over 40,000 patients at the South Department, without a dangerous symptom attributable to antitoxin.

The work of Theobald Smith, followed by the work of Otto, Rosenau and Anderson,[17] proved that horse serum administered to guinea pigs sensitized them, after an elapse of about ten days, to the same kind of serum. A small second dose was often sufficient to cause death with symptoms of distressed respiration, cyanosis, convulsion and finally death. This phenomenon has been termed anaphylaxis. While this reaction is common in laboratory experiments with guinea pigs, it is very rare clinically. It would be a great pity if these interesting laboratory experiments should curtail the use of antitoxin in diphtheria. It has been thought that the rare appearance of severe symptoms following the use of the first antitoxin dose is due to a natural "sensitization," or anaphylactic condition. The symptoms are marked respiratory distress, and may be due to the contraction of the smaller bronchi. Asthmatics, hay fever patients, and those sensitive to the emanations from the horse are more liable to have symptoms of anaphylaxis on administration of antitoxin. Adults seem to be more subject to this condition than young children. It would seem, if such patients require antitoxin, that they should be given a preliminary dose of atropin, followed by a tentative dose of antitoxin. The writer resorted to this procedure on one occasion where the patient had experienced severe respiratory disturbance following a dose of antitoxin in a previous attack of diphtheria. The patient was given a full dose of atropin, hypodermatically, just fifteen minutes prior to the antitoxin.

Concentrated antitoxin was then administered in small doses every fifteen minutes until the required amount was given. In patients in whom there is a suggestion of a sensitized state, this method is worthy of a trial. It may be of interest to analyze the sixteen deaths reported in the literature following the use of antitoxin. The size of the dose in each case was small, varying from 500 to 4,000 units. Only six of the sixteen cases were suffering from diphtheria, and six others, owing to exposure to diphtheria, were given small immunizing doses. The remaining four did not have diphtheria at all, but were given antitoxin for asthma. Nine of the sixteen gave a history of asthma in some form.

There is another condition known as "serum sickness" following at a variable interval the administration of antitoxin, usually about the tenth day, characterized by rashes of various kinds, with intense itching, joint pains and fever. There may be some edema. The pulse is quickened, but of good strength. This condition may at times be mistaken for measles or scarlatina, though there are not the usual symptoms of these diseases. Park says that about 20 per cent develop the rash. The reaction is certainly less in the concentrated serum where the irritating proteids have been removed by the Gibson process than in the more bulky product. Calcium chlorid[18] has been strongly recommended in gram doses, beginning on the day of the injection of antitoxin.

The treatment of serum sickness is largely symptomatic. Sponge with cold water for the fever, menthol solution for the itching, and heat to the swollen joints.

The general treatment of a patient with diphtheria should be the same as other acute infectious diseases. The room should have abundance of fresh air. The food should be light and nourishing. The urine should be examined occasionally. On account of the heart bearing the brunt of the attack, it should be guarded as much as possible.

The patient must be kept quiet in bed, not only during the attack, but for a considerable period of time after the illness, depending somewhat upon the severity of the attack. The time should also be longer in cases where antitoxin was given late.

Cardiac stimulants should be given as the heart shows signs of weakness. This can best be determined by listening to the

heart sounds, particularly the second pulmonic. Jacobi[19] has always been a great advocate of alcohol in some form. At the South Department, Boston, reliance is placed on camphor, caffein, atropin and strychnin.

Cracked ice held in the mouth is grateful, and ice packs to the neck are useful, particularly where edema is present.

The use of local antiseptics is of secondary importance, and if strong may be actually harmful. In my judgment, anything stronger in the nose than normal salt solution or liquid vaselin is not to be recommended. To the pharynx in young children antiseptics are of doubtful value, as the physical resistance offered may be more harmful than the local treatment is useful. In older children and adults, my favorite application is a solution of one part of peroxid of hydrogen to three or four of water. Some advise Loeffler's solution, while others prefer irrigation with a hot normal saline solution. The ear should be watched for a beginning otitis media.

Paracentesis should be performed early. Borden[20] lays stress on the number of mastoid involvements discovered at autopsy in patients dying with diphtheria whose drum membranes were apparently negative.

Pneumonia is a serious complication, and is especially frequent in laryngeal cases. Stimulants should be given as the case requires.

Gallop rhythm is a serious complication or sequela, and is best treated by absolute rest, rectal feeding, and appropriate stimulation.

Diphtheritic paralysis is a frequent sequela of diphtheria. It may attack any nerve, but particularly those of the palate and larynx. It is more serious when the pneumogastric is involved.

In laryngeal diphtheria, particularly in intubation and tracheotomy cases, it is advisable to use steam inhalations under an improvised tent. Ipecac may be used in slight stenosis pending the action of antitoxin. This should be relied upon but for a brief period of time, for we must remember that the laryngeal stenosis not only taxes the respiratory system, but gives an added burden to a heart already weakened by the diphtheritic toxin. As before mentioned, these are cases requiring large doses of antitoxin.

In intubation cases the question of feeding comes up. As

a rule semisolid food is best, and should, if possible, be given in the upright position. Rarely is it necessary to use a stomach tube or the Casselberry position.

Whether intubation or tracheotomy is preferable in laryngeal stenosis of diphtheria has long been a mooted question. Since the introduction of the intubation tubes by O'Dwyer, of New York, which marks one of the brightest spots in American medicine, physicians on this side of the waters have shown a decided preference for intubation. France has for a long time been an advocate of intubation, while Germany is using more and more the O'Dwyer tubes. England, in her conservatism, clings to tracheotomy. It is not to be understood that intubation is to entirely supplant tracheotomy, for there are certain conditions where tracheotomy is preferable.

The advantages of intubation are:

1. Relief of the stenosis without the shock of a cutting operation, and without an anesthetic.

2. Consent of the family for operative relief is more readily obtained. This is a great advantage, as early relief of the stenosis is a great factor in reducing the mortality.

3. There is no danger of local infection, and less danger of pneumonia in intubation than in tracheotomy.

4. There is no resulting scar.

Tracheotomy instruments should always be at hand where intubation is performed, in case of stoppage of respiration from pushing the membrane in front of the tube. This is not a frequent occurrence. Rarely the patient persists in coughing up the tube, which, if continued, requires tracheotomy. There are also cases where there is marked pharyngeal swelling, together with edema of the aryepiglottic folds, where tracheotomy is indicated. Lastly, if the surgeon cannot remain within easy reach of the patient, in case of stoppage of the intubation tube, or its coughing up, it might be safer to perform tracheotomy. It does not seem necessary to give the technic of intubation or tracheotomy, for excellent descriptions of these operations are given in our textbooks. However, the ingenious method of Mosher's[21] intubation deserves mention, and in the hands of some should prove simpler than the indirect method. The same O'Dwyer tubes are used, only it is performed with a straight introducer, through a laryngeal speculum. If the direct method is used for diagnosis, it might

be best to continue the operation and insert the tube with Mosher's introducer.

The writer wishes to emphasize the following points:

1. The diagnosis of diphtheria depends primarily on finding the organism. Cultures should be taken of every sore throat in children, and likewise suspicious nasal discharges. But in the absence of a positive finding, if the symptoms point to diphtheria, antitoxin should be given. This is particularly important if the symptoms are severe.

2. Much larger doses of antitoxin should be given in diphtheria. Textbooks have been misleading in this respect.

3. Laryngeal cases are serious partly from mechanical obstruction, requiring prompt relief of the stenosis, and large doses of antitoxin. Intubation in the main is preferable to tracheotomy. Laryngologists should perfect their technic in this operation until it becomes a fairly simple one.

4. Epidemics of diphtheria are kept up largely by "carriers." They should be sought out, isolated, and the abnormality treated. Diseased tonsils and adenoids may require removal.

REFERENCES.

1. Hiss and Zinser's Bacteriology.
2. Darling: Boston Medical and Surgical Journal, December 14, 1911.
3. Jackson: Laryngoscope. Editorial, February, 1912.
4. McCollom: Osler's Modern Medicine, Vol. II.
5. Place: Boston Medical and Surgical Journal, December 14, 1911.
6. Seligmann: Zeits. f. Hygiene, Vol. XX, 1911, p. 68.
7. Greene: Laryngoscope, August, 1908.
8. Page: Archives of Internal Medicine, January, 1911.
9. Lorenz and Ravenel: Journal A. M. A., January 22, 1912.
10. Editorial, Journal A. M. A., September 14, 1912.
11. Levinson: New York Medical Journal, January 6, 1912.
12. Rosenau and Anderson: Hygienic Bulletin No. 38.
13. Fritz Meyer: Berlin klin. Wochen., June 28, 1909.
14. Eckert: Handbook of Serum Therapy.
15. Hoesch: Deutsch. Med. Woch., 1911, p. 1683.
16. Park: Journal A. M. A., February 17, 1912.
17. Rosenau and Anderson: Hygienic Bulletin No. 29.
18. Nuttall and Graham: Smith, Diphtheria.
19. Jacobi: Treatise on Diphtheria.
20. Borden: Transactions Ninth International Otological Congress.
21. Mosher: Laryngoscope, September, 1910.

LII.

TWO CASES OF PERFORATION OF THE PETROUS PORTION OF THE TEMPORAL BONE DUE TO THE CAPSULATED BACTERIA.

By H. B. Graham, M. D.,

San Francisco.

In the Annals of Otology, Rhinology and Laryngology for December, 1910, I made a short review of the work that had been done on the capsulated bacteria in the acute middle ear infections. Since then I have had the misfortune to operate two cases which illustrate the insidiousness and burrowing power of this group of organisms. I append the two histories and photographs of the temporal bones.

Case 1.—M. D., age 50 years, came to the clinic February 19th, with a history of having had a severe bronchitis in January. About February 5th had had a dull, left-sided, intermittent pain in the head with hissing noises and deafness in the left ear. No discharge. Examination at the clinic gave a right temporal headache, pain over left ear, with a marked external otitis. The patient did not return to the clinic until March 3d, when I saw him for the first time. At this time there was a subperiosteal abscess which probably came from the mastoid. He complained of pain over left eye and of slight dizziness. There was no nystagmus. He was operated March 4th. I uncovered the sinus and dura in posterior and middle fossæ and the whole mastoid was removed. The temperature, both before and after the operation, for two days ranged between 98° and 99° F. On March 7th the patient was feeling well, the pain and dizziness had left, and he was permitted to leave the hospital. On March 8th he was taken to the City and County Hospital unconscious, with a temperature of 102° and pulse 120. On March 10th exitus.

Autopsy Findings.—(Dr. Ophüls.)—Walls of the mastoid cavity were made up of apparently normal bone. At the base

of the cavity, near middle ear, were a few drops of thick yellow pus; postmortem clots and liquid blood in the longitudinal sinus. Meningitis purulenta at base of brain. Underneath the tentorium, about 1 cm. laterally from internal auditory canal, there is a hyperemic spot in the dura about 1 cm. in diameter with a central grayish discoloration; underneath the dura there is a large ragged opening in the bone about 1 cm. in diameter; there is a small accumulation of pus between the bone and dura; at the bottom of the cavity there is an opening in the bony capsule of the posterior semicircular canal. In the middle ear there is mucus and pus; the mucous membrane is swollen and hyperemic. From the middle ear a fistulous opening passes through the temporal bone and appears on the posterior surface at the spot where the abscess underneath the dura was found. Smears from the middle ear and meninges show a very small diplococcus resembling the pneumococcus.

In this case the initial bronchitis, followed by a period of indefinite pains in the head with symptoms referable to the ear and a low temperature, apparently over a long period of time, would have led to an earlier diagnosis and operation if the patient had not been so irregular in attendance and had been more communicative when in the clinic. That this man carried a meningitis about with him for days before he died or was even operated can hardly be doubted from the appearance of the meninges at autopsy, and still he had but few symptoms that were referable to the meninges at the time of operation. The meningitis due to these capsulated bacteria is an extremely quiet one and can very easily be overlooked by the most careful observer.

Case 2.—A. C., age 49 years, steward, Scotchman. Entered clinic October 10, 1912, complaining of pain over the right ear and extending to the top of the head. There was a large amount of pus in the nose and an ethmoid operation was done. Following this the patient disappeared, and entered the clinic again October 30th, stating that he had had a pneumonia and pleurisy. I saw the patient for the first time on October 30th, when he had an abscess behind the ear. I operated, finding pus under the periosteum and a fistula under the anterior spinous process. The longitudinal sinus was covered with granulations. The temperature was normal after the operation and the patient supposedly made an uneventful recovery, going to

work with a small opening behind the ear. On December 6th the patient was readmitted to the hospital with well-marked signs of a meningitis. Lumbar puncture showed a turbid fluid with shreds. The temperature rose rapidly from 101° to 104°, pulse from 90 to 150, and exitus in December.

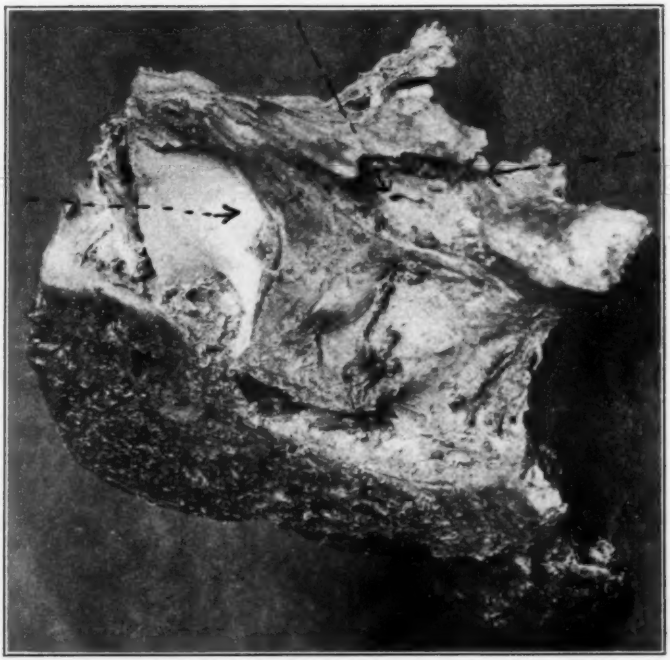
Autopsy Findings.—(Dr. Ophüls.)—Scar from old mastoid operation on left side; opening from a recent operation on the right. The meninges are covered with a thick purulent exudate. On lifting the brain from the petrous portion of the right temporal bone there is seen an opening in the bone at the lateral edge of the temporal ridge about 1 cm. in diameter and one-half cm. deep, which is somewhat crescent shaped. A little cavity is formed by the defect between the dura and the bone, in which there is a small amount of pus. This cavity connects by a fistula with the middle ear at a point on the floor in front of the eustachian orifice. The bone and under surface of the dura are covered with granulation tissue. The middle ear contains a mucopurulent fluid, and pale granulation tissue covers the small bones. The operated area consists of grayish red granulation tissue several millimeters in thickness. There is no pus in the internal auditory canal. Left middle ear normal. Left frontal and right and left ethmoidal sinuses contain a small amount of mucopus. There is a syphilitic atheroma of the aorta. Smears from the wound, meninges, lateral ventricle, middle ear, subdural abscess and lung show many polynuclear leucocytes and many encapsulated diplococci and short streptococci and streptococcus mucosus.

In this case the fistula had taken a course diametrically opposite to that in the former case, the one appearing in the posterior, the other in the middle fossa.

The accompanying illustrations are self-explanatory.

Posterior semicircular canal.

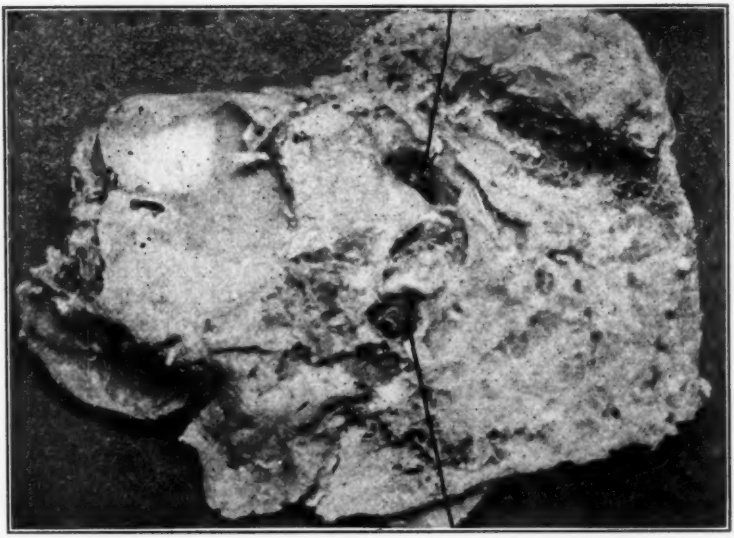
Internal auditory canal.



Fistula from antrum.

CASE 1.

Oval window.



Fistula from middle ear.

CASE 2.

CASE 2.

LIII.

THE SURGICAL MANAGEMENT OF DISEASES OF THE FRONTAL SINUS.

By Robert Cunningham Myles, M. D.,

New York.

The problem as to the best method of surgical procedure in each case of frontal sinus disease has created many anxious moments of serious deliberation for the conscientious surgeon. The cause, process, and pathologic stage are important factors to be considered in our plan of management.

Cases of acute frontal sinusitis with obstructive symptoms should be controlled by strict discipline for the patient, and by prompt, watchful, active, and definite procedures on the part of the attending surgeon. The patient should be ordered to rest, with a minimum of physical exercise. Temporary and repeated applications of solutions of adrenalin chlorid and cocain, used on a small cotton applicator, under the anterior end of the middle turbinal and within the nasofrontal canal, will frequently relieve the ominous and threatening destructive pressure within the sinus. If this fails, the operation for the removal of the anterior end of the middle turbinal should be performed, and drainage of the sinuses secured by the dilatation probe or by removal of a part of the anterior ethmoidal cells and the projecting portion of the nasal process of the superior maxillary bone. The importance of limiting the operative area as much as is consistent with securing sufficient drainage should always be carefully considered, on account of the possibility of spreading the acutely infective process. The writer believes that we would be justified in making a temporary drainage externally above the inner canthus of the eye, in case of failure of attempts to relieve the disease intranasally, because he believes that it is wiser and safer to

delay the performance of the radical operation for about three weeks in order that the usual virulence of the infective bacilli may become exhausted.

The proper management of the acute and subacute cases of frontal sinusitis will lessen the number of chronic cases by a large percentage. There are many classes of types of cases of chronic frontal sinusitis demanding a nicety of individual judgment, and when we consider the responsibility assumed by the authority of our judgment, we are well aware of the hazard encountered in making statements as to rules for definite methods of procedure. Our science has not advanced far enough to warrant unerring rules of accuracy in all cases, but it is with pride that we notice the wonderful improvement which has been made in the treatment of the diseases and in the operative technic since the early days when the writer was so actively struggling in the mystic meshes of these complex cases.

It is evident that the pendulum swings too far with all of us in regard to the internal or the external operation, and it is also evident that habit or bias influences our methods of procedure when a cold analysis of symptoms and physical conditions should determine the best plan of surgical management. Very serious conditions often require desperate remedies. We should be prepared to do a complete radical operation when there is considerable necrosis of the mucous membrane, periosteum, or of the bone, with or without perforation of any of the walls. When there is a neoplastic growth, especially of a malignant nature, an external radical operation is necessary, though there may be an exception in some cases of syphilis. The very small sinuses can be managed by the open method through the floor beneath the orbital ridge, without much facial deformity.

The Killian operation is the best method of procedure in cases of serious disease of the large sinuses, especially where previous injudicious and incomplete operations have been performed. Sometimes a partial Killian, using his bridge with the open method, will afford the best results in certain types of cases of the latter class. Since the writer many years ago adopted the habit of removing the anterior end of the middle turbinal, most of the ethmoid cells, and a part of the nasal process of the superior maxillary bone, he has "managed him-

self out of" an opportunity to operate externally in over 90 per cent of his cases.

The use of drainage tubes from the sinus to the nose has been abandoned after the performance of internal operation, because they seemed to excite that granulation tissue which is nearly always the cause of the failure in our unsuccessful cases. The writer has always condemned semi- or thorough curettage of the sinus where we do not do the complete obliteration operation. Free drainage and free ventilation are far better for an edematous and infiltrated mucosa than a surgical mutilation. Irrigations with normal salt solution or a solution of boric acid, passed up through the enlarged naso-frontal duct with the author's malleable silver canula, have proved to be of valuable service in bringing about a favorable result. Filling the cavity with solutions of argyrol, iodoform or vaselin, or Beck's paste has, at times, apparently overcome very stubborn pathologic conditions.

In conclusion, the writer would advocate certain rules for the surgical management of the diseases of the frontal sinus.

1. When the acute cases have unbearable, severe, and continuous pain in the sinus and above the eyes, attended with profound constitutional symptoms—fever, chills, and depression—we should proceed in a definite manner to relieve the pressure, if necessary, by doing a submucous operation on an obstructive deflected septum and by removal of the anterior end of the middle turbinal, a part of the anterior ethmoidal cells, and the nasal process of the superior maxiliary bone; and, if this fails, should make a small incision in the skin above the inner canthus of the eye and a small opening in the bone—a radical operation to follow a few weeks later, if necessary.

2. Chronic cases should be treated by the external operation where there is evidence of bone necrosis, either with or without perforation; for neoplasm, especially of malignant nature; for meningeal symptoms which are probably due to sinus disease; for continued attacks of severe pain and discharge mixed with pus and decaying débris; and to satisfy patients who demand relief from recurring attacks of frontal sinusitis after failure to obtain relief by the internal operation.

The Killian operation, combined with removal of the anterior ethmoidal cells and nasal process of the superior max-

illary bone, has proved to be a most satisfactory method in cases of large sinuses. The drainway into the nose from the space where the sinus had been should be kept open by tubes until the open space under the bridge has become obliterated. Compression over the sinus will aid materially in hastening the healing. The use of the artificial blood clot—either combined with paraffin or not—will decidedly relieve many of the objectionable deformities.

LIV.

REPORT OF A CASE OF ULCERATION OF THE LARYNX; PERICHONDRITIS OF THE ARYTENOID CARTILAGES, ABSCESS AND PARTIAL EXFOLIATION OF BOTH CARTILAGES RESULTING FROM TYPHOID FEVER.*

By J. H. Bryan, M. D.,

Washington, D. C.

This case is reported in order to emphasize the importance of making regular and systematic examinations of the upper air passages, especially of the larynx, in all cases of typhoid fever, in order to detect the early changes that take place in the mucous membranes of the upper air passages in this disease.

On the night of October 12, 1912, I was called to see Major C., U. S. A., aged forty years, at the Walter Reed Hospital, suffering from a severe case of typhoid fever, then in about the fiftieth day of the disease. I was called to see the patient because of his difficulty in breathing and loss of voice. The following history was given me by the attending surgeon: In September this officer returned to his duties in Washington, after a western trip, suffering from typhoid fever. The disease ran rather a severe course from the beginning, with high temperature and several severe intestinal hemorrhages. The temperature gradually subsided, and within a week or ten days of my seeing the case he was supposed to be convalescing. About four days before I was called to see him, the patient was thought to have taken cold, a slight laryngitis developed, accompanied by a cough and a husky voice, but at no time did he complain of any pain in the region of the larynx.

When I saw the patient, he presented a very emaciated appearance, with an anxious expression of the face, skin bathed

*Read before the American Laryngological Association, Washington, May, 1913.

with sweat, respiration rapid and shallow, temperature 100°, pulse small, thready, and rapid, and voice husky. Laryngoscopic examination showed fauces very much congested, but no ulceration in this region. There was extensive swelling of the right ventricular band, which overlapped and partially obscured the corresponding true vocal cord, which was in the median line and did not move on deep inspiration; there was also seen a deep ulceration on the under surface of the ventricular band, which seemed to extend to the free margin of the true vocal cord. Both arytenoid cartilages were markedly swollen and posteriorly were bathed in a thin, grayish secretion, probably purulent in character. The left vocal cord moved somewhat on inspiration, leaving a narrowed glottis, sufficient, however, to admit of respiration.

The gravity of the situation was explained to the family and a tracheotomy advised, but owing to the fact that he seemed to be breathing deeper and was more quiet, I left him about midnight, cautioning the surgeon in charge to do an immediate tracheotomy if his respiratory difficulty increased. I saw the case again the next morning at ten o'clock, and while the patient had passed a restless night, he seemed, on the whole, somewhat improved as to his local condition, but not so well generally. The examination of his larynx showed the condition about the same as when last seen at midnight, with the exception that the excursion of the left vocal cord was greater, giving a much wider chink to the glottis and thereby admitting of freer respiration. The consent of the family having been obtained, it was arranged that I should return to the hospital at two o'clock, when a tracheotomy would be done. I had not been away from the hospital more than an hour when the patient had a severe coughing spell which brought about a spasm of the larynx, necessitating an immediate tracheotomy, which was done by Captain Smith, the medical officer in charge of the case. The patient did not breathe after the trachea was opened, and died almost immediately, although stimulation and artificial respiration were resorted to. Owing to the prolonged illness, the patient's heart was greatly weakened, and his death was due to heart failure about the time the trachea was opened.

The larynx was removed, and unfortunately placed in Müller's fluid, which discolored the specimen. The following day

Major Russell of the Army Medical School and I opened the larynx and found the following conditions: The right ventricular band swollen, and a large ulceration on its under surface; the true vocal cord ulcerated on its free margin; both arytenoid cartilages completely detached and lying loose in their capsules, the right cartilage being held in position by only a small membranous attachment. A bacteriologic examination made by Major Russell proved negative.

PHOTOGRAPH OF SPECIMEN.

The photograph of the specimen does not show the destructive changes that were found in the larynx as clearly as the natural specimen—the shadow is so deep that the right arytenoid cartilage is seen with some difficulty—but it is sufficiently distinct to enable us to see the destructive changes that have taken place.

In the light of what has been accomplished by antityphoid vaccination in the United States army, it may be asked why this officer had not been immunized. Several years ago he contracted pulmonary tuberculosis, and for a while was under treatment for this disease at Fort Bayard, but later was discharged cured and returned to duty in Washington. In view of his previous tubercular history, and as tuberculosis is a well recognized contraindication to typhoid vaccination, it was deemed unwise to subject him to this treatment.

This brings up a very interesting point in connection with typhoid prophylaxis, and that is, that typhoid vaccine acts very much in the same way as tuberculin, with a constitutional reaction and fever lasting several days. Some French surgeons claim to have had cases in which latent tubercular lesions have been excited and made quite evident after typhoid vaccination.

Owing to the insidious character of the development of this complication, it is wise that a laryngeal examination should be frequently made of typhoid cases, especially so when there is the slightest change noted in the character of the voice, cough, or difficulty in respiration. Jackson,[1] in a very interesting article on this subject, after a study of 360 cases, quotes Dr. John W. Boyce on this point, who says it is a question of great interest whether it is possible for these affections to cause a fatal termination without such symptoms as shall plainly indicate the nature of the occurrence. He further

says, theoretically it seems impossible that a man should choke to death without the conspicuous symptoms of asphyxia, particularly cyanosis, but observation has convinced him that it is only too true; that these cases may sink away as peacefully as a baby goes to sleep, and that one may not in the least suspect what is happening.

Jackson divided his cases into subacute catarrhal laryngitis, ulcerative laryngitis, and perichondritis. The first was found to be present in 227 cases (or 63 per cent), the second in 68, and the third in 17, of the 360 cases.

St. Clair Thompson,[2] in the chapter on laryngeal complications in typhoid fever, in his work on "Diseases of the Nose and Throat," gives the frequency of occurrence from the European standpoint as follows: Landgraf, 11 per cent of all fatal cases; Griesinger, 26 per cent of all fatal cases; Kanthack, 26 per cent of all fatal cases; Ouskow, 30 per cent of all fatal cases.

It is difficult to arrive at any conclusion as regards the comparative frequency of this complication of typhoid fever in this country and abroad. The figures given by Jackson seem to show that a much larger number of cases of laryngeal involvement occur in this country than is indicated by the figures given by Thompson. The epidemic in which Jackson made his observations, however, was an unusually severe one, and his subjects were largely of a poorly nourished type, and this may account for the apparently greater frequency of this complication in this country.

We will not be able to get at the truth of this matter until more careful observations are made, not only in the hospitals, but in private practice as well.

REFERENCES.

1. Jackson: Journal American Medical Association, November, 1905, page 845.
2. St. Clair Thompson: Diseases of Nose and Throat, page 671.

LV.

VASOMOTOR DISTURBANCES OF THE UPPER AIR TRACT.

By C. W. Richardson, M. D.,

Washington.

During the past few years it has been my observation that the rhinologist is beginning to appreciate the fact that the upper air tract, especially the nasal tract, is not an independent organ of the body, but that it is part of an organism and is influenced pathologically, to a large extent, by disturbances in other organs and systems of the body. Acting upon this gradually increasing impression among rhinologists, members of our specialty are looking, or beginning to look, beyond the nose to determine the cause of and remove the conditions which bring about various diseased activities within the nasal chambers. One is impressed with the increased frequency with which very excellent and timely papers are appearing in the journals bearing upon and elucidating points treating upon this corelationship of the nasal organ and the bodily condition, evidenced by thought, research and keen study, often with reports of cases bearing upon some important type of interrelationship. My only excuse for reciting the few words I shall bring to you is that the subject is such an important one that it should be furthered in every way that our feeble insistence can carry it. One is impressed with the decreasing frequency with which intranasal operative work is now being done for vasomotor disturbances of the upper air tract. Some years ago it was the common procedure to cauterize and remove turbinates for this disturbance. I regret to note that, even at the present day, this type of surgery is too much indulged in in efforts to relieve this condition. It has been my experience that this form of nasal disorder, if thoroughly studied from its general aspect and those conditions which are instrumental in provoking it, can be relieved

or removed and a moderate degree of simple nasal treatment instituted, in which surgery plays no part, a resolution with permanent return to the normal can be assured.

Only a few months ago I discharged from my care a case exemplifying this condition, upon which a submucous operation for restoration of a deflected septum had been done. The operation, as far as I could see, had been attended with a perfect operative result, but the patient had never received any beenfit so far as his breathing was concerned. The septum was perfectly straight, but the sodden, swollen turbinates filled in all the breathing space. Proper instruction as to diet, exercise and avoidance of overwork and excessive nervous zeal, with mild local treatment, resulted in a rapid improvement and later a complete restoration of the normal lumen of the nasal cavities.

Another type of case which I fear also the rhinologist has too lightly considered in the past is the large group of cases wherein there is more active congestion of the turbinates, with intermittent turgescence of varying degrees. Close inquiry in many of these cases will demonstrate that the disturbance is brought about and maintained by faulty metabolism and autointoxication. All methods of local treatment of a surgical character employed to relieve such conditions, independent of a proper appreciation of their general causation, are attended with only temporary relief. It is only through a careful consideration of these cases from the gastrointestinal aspect, and the application of the proper therapeutic treatment along these lines, that a permanent resolution can be hoped for and maintained. How often are the middle, and even the inferior, turbinates more or less sacrificed in the attempts to relieve these conditions, when the result could have been more certainly, rationally, and, for the well being of the patient, more satisfactorily accomplished by properly applied therapeutic measures.

Another series of cases which claim our attention and that are provocative of disturbance within the upper air tract are those produced by errors in diet. It has been my observation that investigation of the diet of patients will often lead us to ascertain the cause of many cases of turgescence of the turbinates and various reflex disturbances in the oral and nasopharynx. In many such cases we will find the patients are

partaking too largely of carbohydrates and sugar. Such patients are usually overfed, improperly clad, and do not take the requisite amount of physical exercise. The proper regulation of the diet and due regard to the habits of living will aid us very materially in bringing about restoration to the normal in this class of cases. Besides these cardinal general conditions cited, there no doubt occur to many of you others that could be brought forward wherein the chemistry of food, the biochemic action of various secretions, and the development of toxins within the body excite these deleterious actions directly or indirectly upon what is—for want of a better term—designated the vasomotor system of the respiratory tract.

It is only my intention in this short paper, in a general way, to aid in impressing again the important fact that there are many disturbances of the general system which influence the upper air tract from a pathologic point of view, and the relationship of these to the disturbances noted must be thoroughly weighed before any operative measure is resorted to; otherwise, disappointment may not only be keenly felt by the patient, but also by the operator, as the result of illmeasured intranasal operative efforts.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of April 11, 1913.

Paper: The Modus Operandi of Galvanic Nystagmus.*

By Lester Mead Hubby, M. D.

DISCUSSION.

Dr. Phillips said that he had had the pleasure of reading Dr. Hubby's paper, and had already congratulated him on the very effective work done in presenting a clear and lucid explanation of the action of galvanic irritation upon the semicircular canals. The diseases of the labyrinth are of great interest to all otologists, and it was necessary only to look over the programs of the section for the past year to note how much the minds of the members are turning to this topic, and to see how much has been accomplished in this line. Notwithstanding all this, we are still very much in the dark regarding the practical application of the knowledge possessed, which is necessarily still incomplete. The work which is being done, however, and the various papers which are being presented are helping to settle many questions. Dr. Phillips said that he had not himself given this phase of the subject the time and attention which Dr. Hubby had devoted to it, and therefore hardly felt competent to discuss it. Dr. Phillips said that Dr. Hubby had done most of this work in his service, and expressed the hope that other of the younger men would continue work along this line.

Dr. Isidore Friesner said that all the members of the section should be grateful to Dr. Hubby for his excellent paper. There

*See page 638.

are no other tests of ear conditions that are fraught with so much of discomfort to the physician as well as to the patient, and the utmost patience on the part of both is required. The results of galvanic tests are so surprising in their lack of uniformity that it seems impossible to evolve a classical picture. In suppurative labyrinthine disease the galvanic test gives us little or no information. We know from the experiments of Marx and from the examinations of Neumann and Ruttin that there is a reaction to galvanism long after the labyrinth is destroyed. He was surprised in noting Dr. Hubby's work to observe the effect of the galvanic current upon the auditory apparatus, at times increasing and again abolishing tinnitus. That seems to be an exceedingly fruitful field of study. There are many socalled cases of chronic catarrhal otitis where, in all probability, we are dealing primarily with neuritides and secondarily with atrophic changes in the end organs, and since observing Dr. Hubby's work he has wondered whether the galvanic test would not offer some help in differentiating these conditions.

Dr. Ballin said that the subject of galvanic nystagmus as presented by Dr. Hubby was of great interest, but it is to be regretted that the consideration of a subject like this leaves us still very much in the dark, for all such papers are necessarily based upon theory and not upon practical experiments.

One of the most carefully prepared papers on this subject that he had seen was published by Brünings, dealing with the cataphoresis theory, and that the flow of the stream of endolymph is affected by the application of the anode and cathode to the mastoid region. You get a flow of the endolymph in one direction upon the application of the cathode, and the reverse when the anode is applied. This theory does not seem to be generally recognized, however. Marx of Heidelberg carried on a series of experiments on cats. In some of the animals he filled the labyrinth with lead, while in others he destroyed the labyrinth entirely. Upon applying the galvanic current he found that there was still a reaction in the form of a perceptible nystagmus. These tests tend to show that the galvanic nystagmus is due to irritation of the nerve endings, and, as Dr. Hubby says, to the nerve endings in the cristæ. The nystagmus produced by the caloric and rotatory tests are, however, the most reliable. It is found, however, that when

the labyrinthine structures were destroyed by pus, etc., a galvanic reaction could still be obtained. According to Neumann, when he did not get this, he claimed that its absence was due to a retrolabyrinthine condition, and that he could thereby detect whether the lesion was located in the retrolabyrinthine or in the labyrinth itself. Of course, this is all theory, but Dr. Hubby's paper had certainly awakened thought for further investigation.

Dr. McCoy also congratulated Dr. Hubby upon his interesting paper. Such papers have both a theoretical and a practical value. We cannot write or talk too much about the labyrinth at present, for all the knowledge concerning it seems to be confined to the otologists. It is surprising to note how very little most general physicians seem to know about such conditions. He had recently been called in consultation with a physician whose opinion he values highly on most topics. This physician had asked him to open the ear drum of a woman patient, which he did. The physician said that he would look after the case, and five days later he stated to Dr. McCoy that this patient had tried to get up in the middle of the night, and was seized with a vertigo, and felt as if she were being turned about; she returned to bed and waited in this condition till morning for the physician, and he said she had an attack of hysteria. She was having some trouble with her husband. He treated her, and then asked Dr. McCoy to see her two days later. She then had quite a marked spontaneous nystagmus, and yet this physician had no conception of what was going on in the internal ear.

We cannot read or publish too much about the labyrinth, for it makes the subject more widely known and calls attention to it. Dr. McCoy said that he had hoped that Dr. Hubby would make some suggestions as to how to make galvanism more practicable. Up to the present time it has been more or less discarded because of the dictum of Neumann concerning it. The only improvement is that we may differentiate it by the fact that on the side on which the labyrinth is affected we have to use a stronger current to produce nystagmus.

Dr. Hubby said that galvanism makes the nerve more irritable, and in this way we may be able to bring out a spontaneous nystagmus; sometimes we think we have it but we are not sure. The theory of Bárány and that presented by Dr. Hubby are

very interesting. Dr. McCoy said that he would be inclined to accept the view presented by Dr. Hubby. He thought it more logical than the view of either Bárány or Brüning. It seems to cover the field thoroughly and practically.

Dr. Fowler said that it was practically impossible, even for one who had looked into the subject quite carefully, to follow the spoken argument of a paper like Dr. Hubby's. The facts and the references to the labyrinth come with such rapidity that one cannot keep in mind exactly what the speaker is saying, and therefore it would be useless to attempt to discuss many of the points which suggest themselves, but by making a diagram upon the blackboard he thought he would be able to present one or two more points simply and clearly.

Dr. Hubby had spoken of the impossibility of electrically stimulating the semicircular canals individually. The same difficulty enters into every test method that we have today. Any fluid current in one must necessarily cause a current in another canal, either by pressure or siphonage, the latter especially in the two vertical canals, on account of their joining. A like way with the horizontal canals, etc.; the disturbance may not cause an actual current, but a tendency to change in the movement of the current in some degree. If we bear in mind the exact relationship of the orifices of the canals to each other, light will be thrown upon these tests and explain why they are not more accurate. The wonderful thing is that they are as accurate as they have been. He had made measurements on the actual bones of all of these parts, and these explain certain results obtained in practice from rotation, but it would be impossible for the members to follow the data off hand. The hydraulic interrelation of these canals should also be considered, although it may be that is not the important factor in their action. The placing of the maculæ of the canals is very suggestive. It can be easily seen that they may be affected by pressures in or of the endolymph, due to the centrifuging or flow of this fluid, whereas the maculæ of the utricle and saccule are so placed that they may be influenced by the faintest changes of pressures, but not by fluid flows. It may be that it is a combination of the endolymph flow in the canals and the pressure in the larger sacs which determines the labyrinthine influences.

Dr. George E. Davis agreed with Dr. McCoy, that the whole subject was difficult and not well understood by the general practitioners, indeed not by all specialists.

Dr. Hubby's explanation of the mechanism of the reflex of galvanic nystagmus was most interesting if not entirely convincing. It must be borne in mind that nystagmus is a reflex produced by some stimulation or irritation in various ways—mechanically, chemically and electrically. The different tests employed to elicit this impulse may be explained on different lines.

(1) Mechanically, by the stress, pressure or momentum of the endolymph current produced: (a) by rotation; (b) by air pressure or rarification, if a fistula be present; (c) calorically by hot or cold water.

(2) Chemically, by the application of irritants directly to the end organs of the static labyrinth.

(3) Electrically, by the application of the galvanic current.

Different theories have been offered in explanation of the galvanic as well as the other reflexes. Neumann claims that the mechanism of the galvanic reflex and the caloric reflex is more or less similar. Heat and the cathode or negative electrode have a stimulating effect on the end organs of the labyrinth, whereas cold and the anode or positive electrode have a sedative effect.

Another theory of the galvanic test is that it establishes an endolymph movement by kataphoresis and thus produces nystagmus mechanically, as in the rotation and caloric tests. However, it does not seem to him possible to explain the galvanic nystagmus reflex on the mechanical theory, for the reason that you cannot limit or circumscribe the effect of the galvanism on any particular crista, and, moreover, the current affects the nerve trunk as well as the nerve end organ, for it is well known that nystagmus may be elicited galvanically, even after the destruction of the labyrinth. Moreover, the galvanic test is not practical for two reasons: in the first place, it is not accurate, and in the second place, it is very painful to the patient.

Dr. Hubby, in closing the discussion, said that nearly all who had discussed his paper had spoken of the impracticable side of the galvanic tests, and he agreed with most of what had been said.

In many patients one can get no nystagmus whatever with the amount of current that can be used. Therefore the sug-

gestion that it be used as a differential test between labyrinthine and retrolabyrinthine disease is of no value. A step in the right direction, although considered by many a plaything, is the nystagmograph of Buys of Brussels. He claims that with his little capsule which he puts over the eye he can detect nystagmus when using only a slight current. The apparatus, however, is cumbersome and expensive.

He had not spoken of the cochlear nerve tests, for he thought he had too much extraneous matter in the paper as it was.

Brüning's theory that galvanic stimulation of the vestibular nerve was produced by a movement of the endolymph was difficult to harmonize with the fact that with galvanism you can produce nystagmus whether the endolymph is there or whether the canals are all destroyed.

He did not wish to give the impression that he considered irritation of the crista in the rotational and caloric tests was due solely to pressure. But he thought it must be some combination of variations in pressure, torsion, and stretching of the hair cells.

He also thought that the combined hair cells of a crista were in a state of either hyperirritation, normal irritation, or hypoirritation.

DR. GLOGAU asked if Dr. Hubby had made experiments on animals, to which Dr. Hubby replied in the negative. Dr. Glogau rejoined that he merely wished to know what experiments had enabled him to arrive at his theory.

DR. HUBBY replied that it rested on the work of himself and others in trying to find some explanation which would fit all the known facts. It was purely theorizing.

DR. GLOGAU asked whether Dr. Hubby thought this was somewhat different from the others. He would like to have the theory tested in a series of experiments on either people or animals, and then try the conclusions.

DR. HUBBY replied that we know certain facts, and we try to find a theory to fit these facts. That was all that could be said. You can experiment on animals or on persons and get certain results, and then try to explain the results. Previous theories did not seem to explain the phenomenon, and he had tried to find something that would. Of course the exact mechanism of the stimulation of the hair cells in the crista in movements of the endolymph, etc., is still in doubt.

NEW YORK ACADEMY OF MEDICINE,

SECTION ON OTOLOGY.

Meeting of May 12, 1913.

SYMPOSIUM ON RADIOGRAPHY OF THE MASTOID.

Exhibition of Radiograms Illustrating the Pathology of Mastoiditis.*

BY HENRY S. BIRKETT, M. D.

Radiographic Findings Illustrating the Anatomic Development of the Mastoid Bone. (Stereopticon Demonstration.)†

BY WM. H. STEWART, M. D.

Radiography as an Aid in the Diagnosis of Mastoid Disease.‡

BY FREDERICK M. LAW, M. D.

DISCUSSION.

DR. PHILLIPS expressed his regret that he had not been able to arrive sooner, and had seen only the last of the series of pictures presented by Dr. Law. He had seen Dr. Birkett's pictures at the International Otological Congress, and Dr. Birkett had recently asked him to express to the section his regrets that the change of dates had prevented him from the pleasure of being present, and furthermore, to say for him that he still held the views expressed at the International Congress in Boston on the subject of radiography in connection with mastoiditis—that while valuable as a diagnostic aid, he does not allow his clinical judgment to be biased by it.

Dr. Phillips said that he was familiar with most of Dr. Law's pictures. The one showing the septic condition of the lateral sinus was the only one which had been shown at the International Congress in Boston, and, so far as he knew, it

*See page 649. †See page 677. ‡See page 635.

was the only one of the kind in existence—i. e., showing involvement of the lateral sinus. He understood that the findings showed that it was really filled with pus and not a clot. It was very largely pus, if he remembered correctly.

Two or three points in connection with radiography as an aid to diagnosis in mastoiditis are worthy of emphasis. First, the comparison of the plates. Having the two mastoid processes taken at the same time, with an equal exposure. Thus the same relative positions are given, and it is by the comparison of the two plates that the facts desired are brought out.

It is an anatomic fact that the mastoid processes are almost invariably symmetrical. The number, size, and extent of the zygomatic cells are the same, and if there is a large tip on one side, a similar one exists on the other. There are few exceptions to this rule. When one understands the interpretation of these plates as Dr. Law does, and as do some others who are not specialists in such work, the radiographs are an aid in confirming the diagnosis.

Another point is that it usually shows the position of the lateral sinus, and the operator thus has a fairly intelligent idea of the location beforehand.

A third advantage is that there are many times when the otologist feels that a certain case should be submitted to operation, but the family and sometimes the attending physician are averse to it. They are inclined to say: "No; I have seen cases just as bad get well." If, however, you have a radiograph showing clearly the difference between the two sides, to show to the physician or some member of the family, it is most convincing.

Another point which should go on record in discussions in regard to radiography of the mastoid process is that one should be able to make a diagnosis without it, and that we should not permit ourselves to rely upon a radiogram for a diagnosis of an acute purulent involvement of the mastoid; but it is of value as confirmatory evidence, and as such we should give it a place in modern otology. That is the view which Dr. Birkett also holds.

Dr. Freudenthal said that after hearing the interesting papers which had been presented and seeing the excellent pictures which had been shown, he felt he had little to add, especially as it had been only a comparatively short time since he

had been having radiographic pictures made in mastoid affections. He had at home a half dozen pictures which he had intended to bring to the meeting, but seeing the great number of prominent radiologists on the program he had decided not to do so. He could only judge of their value in mastoid affections when compared with accessory sinus affections. While he has several hundred pictures of accessory sinuses, and a much smaller number of the other affections, and while he would not like to operate in many cases of frontal sinus affections without the aid of a good radiographic picture, he agreed with Dr. Birkett and Dr. Phillips that in the majority of cases of mastoid disease the clinical symptoms are so definite that we can very well get along without the aid of a picture. Perhaps, however, after using these pictures for a few years, he may have the same opinion in regard to pictures of the mastoid that he now holds in regard to pictures of the accessory sinuses.

Dr. Hirsch said that he thought credit should be given to the roentgenologist, because he has contributed certain additional data which will assist the otologists in determining both the indication and plan for the operation. Though this work is now almost entirely in the hands of the roentgenologist, the time is not far distant when the clinician, having been taught by the roentgenologist both the technic and interpretation, will himself employ this method of examination as he uses other diagnostic aids. The new tendency is visible in other lines of work in which roentgenology has contributed its quota of diagnostic data.

Dr. Judson Quimby said it seemed to him that the otologist is so able to make a clear-cut finding in regard to the mastoid, that there is very little left for the roentgenologist to do but to confirm his diagnosis. The clinical signs and symptoms of mastoid disease are comparatively clear-cut; whereas the findings in the deeper parts of the nasal sinuses, the ethmoids and sphenoids, are quite difficult. He had found difficulty in convincing otologists that much aid was to be obtained from the roentgenologist. All that he would claim on the mastoid would be to confirm the findings. He rather challenged Dr. Hirsch's statement that he would expect otologists to take up this work for themselves, for it requires a great deal of time, and no busy otologist could give it sufficient

attention to do it well. Dr. Stewart's paper had demonstrated that something more was required than taking a couple of exposures to make a satisfactory mastoid examination. The mastoid should be studied from several different axes. One must have the two sides. If you get the two sides in a normal subject, you can possibly make a comparison, but it is rather difficult. The lateral sinus is especially difficult on account of its variation in the two sides. The right lateral sinus is more spread out and not so clear-cut as the left one. If one will notice a large number of plates, the difference in the two sinuses will be observed. His own technic is about the same as that of Dr. Sidney Lange, of Cincinnati, who has done a great amount of valuable work.

Dr. Sinclair Tousey said that he was probably the first one who suggested the practicability of making X-ray examinations in cases of mastoid disease, having suggested it to Dr. Whiting some nine years ago, but that Dr. Whiting had replied that it was possible to make the diagnosis without it.

The pictures shown by Dr. Law, taken with the patient lying on the back, the occiput lying on the plate, were excellent, but that the same position could be modified by turning the patient's head somewhat to the right for the first plate, and then making a second plate with the head turned somewhat to the left, showing the mastoid cells in their normal position.

The following are some memoranda of the author's methods: There are three principal methods which have proven most useful in his practice. One gives a simultaneous and practically unobstructed view of both mastoid processes. The plate is a large one and is in front of the face, while the tube is in the median line behind the head, at about the level of the occipital protuberance. The mastoid processes are at such a distance from the plate that in order to secure a clear image the X-ray tube should be at a considerable distance from the head, and a diaphragm should be used to exclude secondary rays. There is a very great advantage in having a picture of both sides taken under identical conditions.

The second method is one devised by himself, and has not previously been published. The two sides are radiographed separately, taking great pains to secure symmetrical positions. The plate is at the affected side of the head, which is bowed, and the face turned away from that side; the X-ray comes

from behind and just above the opposite shoulder, passes behind and below the opposite mastoid process, and produces an unobstructed image of the mastoid process, which is almost in contact with the plate. This is to be compared with an identical one made of the other mastoid process.

The third method is one described in his book ("Medical Electricity and Roentgen Rays," W. B. Saunders Co., 1910), in which the X-ray shines through the head horizontally, along a line one inch above the external auditory meatus. This gives a view of the mastoid cells at the level of the tympanum and also a view of the labyrinth.

There are certain general principles which are of prime importance. One is in regard to the use of the fluoroscope. The voice of the tempter tells us that we may safely see enough to enable us to properly adjust the patient and the plate and the X-ray tube. But it is just this kind of work which has caused the suffering and death of so many of the best roentgenologists in every part of the world. It should be absolutely prohibited as dangerous to operator and patient.

A radiograph through any thick portion of the body is always very much better if a diaphragm is employed to cut off secondary rays. And the diaphragm should be made of material opaque to the X-ray but a nonconductor of electricity, so that it may be placed in contact with the wall of the X-ray tube and give a wide angle of radiation.

Intensifying screens may now be had which show practically no grain in the picture and which reduce the time of exposure to one-tenth. I should recommend their routine use for these cases. The same effect is produced by the modern transformer or by the still more recent unfluctuating converter in a fraction of a second as by the old induction coil in a minute or so. And where it may be desirable to make several pictures, the element of X-ray dosage becomes important.

A recent improvement in X-ray generators is the result of a suggestion contained in his book. It is in effect a dynamo producing a direct current of uniform high voltage and an approximately uniform wave-length of X-ray. Regulating the apparatus for a radiograph through the head causes the production to a less extent than ever before of those X-rays which would be arrested and absorbed by the superficial tissues and have a tendency to burn without taking any possible part in the production of the radiograph, because they never

reach the plate. This is a great element of safety in X-ray examinations and also in the treatment of deep seated lesions by these rays.

Dr. Seymour Oppenheimer said that personally he did not think radiography would be of much value in the great majority of cases in the interpretation of disease of the mastoid. In some obscure cases it might be of great value, but they were the exception. Clinical evidence must make our diagnosis and not the radiogram.

One point of great value mentioned by Dr. Law was the recognition of anatomic details. Most operators have at times met with misfortune when chiseling a little too energetically and struck the sigmoid sinus unexpectedly; even when using great caution in keeping close to the posterior bony canal wall, he had at times been mortified to find the sigmoid sinus directly beneath, in a far anterior position, occupying a very superficial position. A radiograph taken as a routine measure would give exact knowledge of the location of the sinus. It would seem to be more valuable for outlining the anatomic details in chronic rather than in the acute type of cases. They would also be of value if a series of radiograms were taken in a case where there is some doubt from the external or subjective manifestations. If the radiograms were taken on a given day and show a slight blurring of the structures, and then another taken several days later showed further blurring, it would be suggestive of an extension of the purulent process. Such an instance represents the value of the radiogram, but not as a general means of establishing a diagnosis.

Dr. Felix Cohn said that he wished to emphasize the point made by Dr. Oppenheimer—that the value of the radiogram at present is not in enabling one to make a diagnosis of mastoiditis, but in giving information in regard to the anatomic details, as we get it in the frontal sinuses, so that we may know beforehand the size and configuration of the sinuses. The point of emphasis is the knowledge of the anatomy before commencing the operation.

Dr. Hubby said that he would like to call attention to one point in the examination of children—especially sick and nervous children. If you examine first one side of the head and then the other, and simply look at the mastoid process without considering the whole picture, you may get an entirely wrong idea of the conditions, for the child is very liable to move,

especially when the diseased side is against the table and plate. If you should find the mastoid on that side blurred and the other side clear, you may come to the conclusion that it is diseased, when the blurring is simply due to a faulty picture. In one or two of the pictures shown by Dr. Stewart it seemed possible that he might have made a misinterpretation of some of the plates. In one case in which he said the character of the cells of the two sides were dissimilar, one plate was blurred while the other side was clear. That always has to be taken under consideration in cases of children. For that reason it would seem advisable to have a single plate taken from the rear, giving a bilateral view, as suggested by Dr. Stewart, as well as the usual side views.

Dr. Stewart is ceratinly to be congratulated on getting such excellent plates in such difficult subjects as young children.

Dr. Van Wagenen said that the first operative case shown by Dr. Law was that of a patient who had been referred by him to Dr. Law with an acute mastoiditis. Immediate operation was contemplated, but he was persuaded to wait on account of the skiagraph. The first plate showed purulent involvement of the aditus. The tip was clear, as were also the cells, whose partitions were sharply defined. The plan of treatment was changed entirely, and operation was delayed for four days. The symptoms then became pronounced. He went in, opened the mastoid, and found the tip and cells free. But a pocket of pus over the sinus compelled him to expose the dura before he got it entirely eradicated. If he had followed his own desire in the matter and operated early, instead of placing so much reliance on the skiagraph, he would have saved at least forty-eight hours and avoided an exposure of the dura.

Dr. Voislawsky said that the plates were valuable in mastoid operations on certain cases where speed was necessary, as in diabetics or cardiacs. If the exact location of the sinuses were known, several minutes might be saved in the operation, to say nothing of the element of safety to the patient.

Dr. Phillips said that he wished to emphasize one fact before the discussion was closed—that there is only one kind of radiograph which is of any value in the interpretation of mastoid cases, namely, a good radiograph. Many of them are poor, and unless the radiograph is a good one and can be contrasted with an equally good one of the other side, it is

not well to rely upon it. At the Manhattan Eye, Ear and Throat Hospital they were extremely fortunate in having a roentgenologist in Dr. Law upon whose work they could rely. Furthermore, he would give full credit to Dr. Caldwell, who was the pioneer in making satisfactory radiographs of the cranium.

Dr. Stewart said that he had little further to add, as most of the discussion did not refer to the development of the mastoid cells. He would like to emphasize, however, that team work was absolutely essential for successful use of the X-ray as an aid in the diagnosis of lesions of the mastoid. The most important value of the radiographic examination was to give accurate anatomic relations in contemplated operative interference. He had stated that it was absolutely necessary to have radiographs of both sides and of about the same density in order to make accurate comparative studies. The radiographic studies that he showed were taken from a number of normal patients in the wards of one of the hospitals with which he is connected. They were taken at random, and the radiographic findings were not selected. He said this in order to show that it is not unusual to find pneumatic cells in children of two, four and five years of age.

He could not agree with Dr. Hirsch, that this work was very simple in its procedure. He had been in busy offices of a number of otologists, and it hardly seemed possible that they would care to take up the work of examining mastoids in addition to their numerous duties.

He expressed regret that Dr. Tousey had not brought some of his radiographs to the meeting, that he might demonstrate the advantages of the methods he advocated.

Dr. Law said that the best illustration he could give of the way in which otologists are taking up radiography was the fact that whereas two years ago at the Manhattan Eye, Ear and Throat Hospital they made plates of possibly one mastoid in two or three months, they now make two or three a day—not so much for diagnostic purposes as for a registration of the anatomy and the location of the lateral sinus. It can be located absolutely and accurately—not as shown in the pictures presented tonight, as they showed the whole structure, taken from an angle, but by taking pictures directly through from one meatus to another or by stereoscopic plates.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of February 18, 1913.

THE PRESIDENT, DR. J. GORDON WILSON, IN THE CHAIR.

Demonstration of Cases.

DR. G. W. BOOT presented the case of Mr. J. McN., aged fifty-four years, janitor by occupation, who, about the middle of December, fell on the ice, striking his head in the region of the right occiput and mastoid. Blood came from the right external auditory canal at this time, and the patient became quite dizzy—so much so that he could hardly keep on the sidewalk when he walked home. He had been more or less dizzy ever since, although the dizziness was gradually passing off. Two days after the accident he had diplopia. This had also passed away. There was at present no spontaneous nystagmus and no unsteadiness in station. Both ears responded to the caloric test. Drum membrane on the right side was thickened; on the left side was about normal. Hearing tests: Bone conduction shortened. Weber (C) to the right. Rinné: right, plus; left, plus. Low limit: right, heard 32; left, heard 32. High limit: right, 9,000, d. v.; left, 19,000, d. v. The upper half of the optic discs was redder than the lower half, and the margin was rather indistinct, but choked disc was not preesnt. Blood pressure, 140.

Exhibition of Specimens.

DR. NORVAL H. PIERCE had very little to say regarding the specimens presented. They were nearly all marked, and were simply specimens of adenoids and tonsils. There was one specimen of adenoma of the larynx, and another of a tumor of the nose, which grew from the region of the sphenoid bone—rather large—in a woman advanced in years, which very closely resembled carcinoma, but inasmuch as there was no recurrence and removal was not especially thorough, the diagnosis of carcinoma could not be sustained. He expected

to find in the collection, which was picked up very hurriedly, a very beautiful specimen of adenoid tissue showing the distribution of the blood vessels, giving a very good idea of the way adenoids undergo involution. The blood vessels supplying adenoids all converge toward a rather limited space, and it is supposed that the connective tissue at a certain time constricts around the blood vessels at the base and thus deprives the rest of the body of the adenoid of its nutrition. He has not found it there, among the specimens. But, as he said, the specimens all speak for themselves.

Pin-Swallowing.

Dr. S. C. Friedberg reported a case of pin-swallowing in a girl of thirteen years, with hysteria. Esophagoscopy was performed.

Relation of Mucoperichondrium and Periosteum of the Nasal Septum.

Dr. Otis H. Maclay said that the specimens showed the relation of the mucoperichondrium and periosteum to each other and to the bony portions. Certain anomalous conditions are present. In one specimen the perpendicular plate of ethmoid occupied almost the entire anterior portion of the septum, leaving only the extreme lower anterior area for a small piece of cartilage.

Discussion of Cases and Specimens.

Dr. Joseph C. Beck said that he had seen the case presented by Dr. Friedberg at the County Hospital, when she first came to him. He got the history that she had swallowed pins. He had a picture made the same evening. In examining the patient he could find nothing in the way of a pin. The girl was typically hysterical, and on questioning the mother it was found that this girl had had a fright by a little boy when she had some pins in her mouth. This fact was not stated by Dr. Friedberg. Possibly this was a factor.

The speaker had a similar case a year ago, which was still more interesting. A girl said she inhaled a pin. He could find nothing, and so had an X-ray picture taken. In the picture a pin showed about an inch away from the soft structures

of the back, over the clavicle. On pressing the girl for a definite answer, she finally said that she knew that it would show in the plate, so kept the pin in her hand and placed it on the plate.

Regarding Dr. Boot's case, he thought there were many things in a case like this that ought to be developed; for instance, the blood pressure, urinalysis, etc., in reference to an arteriosclerosis, which possibly would explain some of these conditions, aside from a pathologic process at the base of the skull. With a pathologic process it seemed to him there should have been more inflammatory conditions.

Dr. George E. Shambaugh called attention to the fact that one of the most common types of malingering is the claim of defective hearing due to accident. It is a common belief that one may assert that he has lost his hearing through an accident and that it is impossible for a physician to discover malingering. He sees a great many railroad cases of this type. As a matter of fact, one usually has very little difficulty in discovering malingering when an injury to the ear is claimed. If there has been an actual injury to the internal ear the symptoms of deafness and tinnitus and vertigo are so characteristic that even when the patient has heard about these symptoms and has not experienced them, he is unable to tell us exactly how they occur. For example, he examined a case a day or two ago. A young man claimed to have been struck by an auto truck and knocked from the steps of a street car. He walked home without any difficulty, and claimed that that night he had a very bad earache, which had kept up ever since. Examination showed the drum membranes were normal. He also stated that he was very dizzy; that he was unable to lie down, and had to get up and walk around. In making the Weber test he insisted that he was unable to hear the tuning fork if he put his finger in the well ear, but could hear distinctly with the ear left open. A history such as this showed clearly the confusion a patient usually has in trying to assume symptoms which he does not experience.

Dr. William L. Ballenger recently had a case of a man who, when riding in an automobile, struck a telephone pole and was unconscious for some days afterwards; he was very giddy and nauseated, and so forth, and he presumed had nystagmus. No observations, however, were made in this

regard. Dr. Ballenger saw the man for the first time three months after the injury, and up to that time he had had some difficulty in walking. All these were phenomena of reaction movements, and the X-ray plate showed a fracture running from one ridge up over the vertex, almost, and around to the opposite supraorbital ridge. It also showed fracture in the base of the skull. Otherwise, he had total loss of hearing in this ear. But apart from this he had made practically a complete recovery. Several X-ray pictures were made of his head at various angles, in order to see if there was any depression of the bone, but there was nothing—simply the fracture without any depression—which accounted for his good condition. The only damage apparently now is his total loss of hearing in the affected ear; the other ear is entirely normal. When the speaker saw him he still had a blood clot in the drumhead of the unaffected ear, but in the affected ear the drumhead was normal. He could not explain this, but simply reported it as part of the case. He thought Dr. Boot's case was one in line. He did not know whether Dr. Boot had plates made or not, but in his case the X-ray showed the fracture very clearly.

Dr. J. Holinger said that of course all were agreed that Dr. Boot's was most probably a case of fracture of the base of the skull. There is a work published in which the writer has given a large compilation of these fractures of the skull and found that the labyrinth and the middle ear are places of predilection for fracture lines. The symptoms must vary according to the parts involved. He had an opportunity of seeing quite a number of these cases in his service at the Alexian Brothers' Hospital. A few years ago there was something like an epidemic of hold-ups by sand-bagging. The hold-up man came upon his victim from behind and hit him with a sand-bag over the ear; the victim would fall down and be unconscious for a longer or shorter period of time. Dr. Holinger saw five or six cases of fracture in cases of this kind, and a number of them were very serious as to their consequences. He observed one man for nine months because of the continued dizziness, which absolutely incapacitated him for work. The moment he tried to stoop he would fall down and, being a laborer, of course that made it impossible for him to find any kind of work. He finally disappeared. No logical treatment was indicated.

Another case, which he had reported to the society before, was that of a man falling from a height of eighteen feet on his head, with consequent hemorrhage from the left ear and unconsciousness, which latter lasted for two or three weeks. Every two or three days he would wake up for a short while and fight violently in bed, trying to take hold of something or somebody. After a little while he would relapse into unconsciousness. He recovered, but there was no hearing in that ear. Dr. Holinger asked him why he fought so violently when he became conscious, and he said when he woke up he would see the room and everything in it normally for a short moment. Soon everything began to turn faster and faster, and he thought that he was falling, and with his utmost power he held himself on whatever he could grasp. The dizziness became worse until he lost consciousness.

The differential diagnosis between fracture and malingering is not very difficult, if you remember the function of the semicircular canals. Often the hearing tests will be of service.

Dr. Alfred Lewy does not think that the diagnosis in an injury case is always so plain. He remembered the case of a man who received a severe head injury in a railway collision. He was seen at his residence about a week afterward. Aside from general nervous symptoms he complained of pain in the ear, noises, and some vertigo. The drum membrane had the appearance of an acute otitis media, and paracentesis was performed. A week afterward the patient reported at Dr. Lewy's office, and tuning-fork tests showed every evidence of a pronounced nerve deafness. Dr. Shambaugh also saw this case and diagnosed nerve deafness. There had been no hemorrhage from the ear, and no organic lesion of the brain was diagnosed. The man was partially invalided for the greater part of two years as the result of his injury.

Dr. J. R. Fletcher thought one thing should be mentioned regarding Dr. Holinger's nine months' case. It seems to him that in a case of this kind, if the labyrinth had not been totally destroyed, the treatment should consist in destroying it. This man was a laborer and could not work because of the disturbance caused by the partially destroyed labyrinth, and it seemed to him that the treatment above suggested would be the best course to pursue.

DR. J. HOLINGER did not know whether he would call the treatment suggested by Dr. Fletcher absolutely good surgery, because we do not know how the fracture lines run, and by operating we may cause a fatal hemorrhage, either subdural or intradural, even at some distance from the place of operation, and unknown to us. The more postmortems one sees of such cases, the more one will be impressed that absolute rest is indicated.

Paper: Topography of the Tympanic Cavity.

BY JOHN A. CAVANAUGH, M. D.

DISCUSSION.

DR. GEORGE E. SHAMBAUGH complimented Dr. Cavanaugh on his beautiful anatomic preparations, and emphasized that everyone who aspired to become a thorough specialist in otology should undertake to work out for himself in this way the anatomy of this region. Anatomy cannot be learned from textbooks or atlases; one must handle the actual specimens in order to get that knowledge which will be of real assistance when working clinically with the ear, and the best way is to make one's own specimens, which will illustrate the various points. In glancing at a specimen of the temporal bone one might suppose that the anatomy of the tympanic cavity could be described in a very few words, but after listening to Dr. Cavanaugh's excellent paper on this subject, one can realize that he has to deal with a very complex anatomic structure. Many of these anatomic facts may at first thought appear to have very little practical interest, but, as a matter of fact, every point in the anatomy of this part has a clinical bearing.

Dr. Shambaugh was interested in the preparation which Dr. Cavanaugh exhibited, showing the aberrant pneumatic spaces extending forward in the petrous portion of the temporal bone. Dr. Shambaugh has a number of specimens of this sort. Infection in these cells cannot be reached by our usual operations.

Dr. Shambaugh called attention to the socalled tympanic cells which develop in the floor of the tympanum. He has several preparations in which these cells extend fully half an

inch from the tympanic cavity. He called attention to the constant pocket just back of the promontory, to which the term sinus tympanicus has been applied.

There was one feature in the anatomy of the tympanum which was not brought out in the part of the paper which Dr. Cavanaugh read that seems to have a very important clinical bearing. This is the duplication of folds of mucous membrane in the cavum tympani. These folds are normally found extending between the fan-shaped ligament arranged horizontally around the neck of the hammer. They frequently separate almost completely the cavum tympani proper from that part of the recessus epitympanicus, to which the term attic is usually applied. Other folds are frequently found connecting the cruræ of the stapes with the niche of the oval window. Another fold is often found connecting the long crus of the incus with the posterior wall of the tympanum. Dr. Shambaugh has a specimen where a fold of this sort divides the antrum, the aditus and the attic into two compartments. These folds of mucous membrane frequently form the basis for those thickened bands that develop in the chronic adhesive forms of middle ear disease. Normally, the folds are made up of two layers of epithelium, between which are located a few capillary vessels. In those individuals where these folds are well developed, either a prolonged middle ear inflammation or repeated attacks of acute inflammation lead sooner or later to the development of connective tissue which produces a marked stiffening in the conducting mechanism. In other individuals, where these folds of mucous membrane are not well developed, there is much less likelihood of permanent disturbances of the hearing resulting from middle ear infection.

Dr. A. H. Andrews heard Dr. Cavanaugh mention the spiculum of bone which frequently extends from the apex of the pyramid across to the inner wall. He has frequently observed that in his studies, but so far has not seen it mentioned in any textbooks on anatomy, and thinks that if it is not mentioned, Dr. Cavanaugh should be given credit for calling attention to it.

Another thing which he thinks should receive more attention is the relation of the facial nerve in its downward course to the posterior wall of the tympanic cavity. He has found

many specimens in which the plate of bone lying between was as thin as bone could well be. Randall mentions two millimeters, but it is frequently very much thinner.

Dr. Shambaugh mentioned the necessity of handling the specimens. The thing that has been of great value to him in the study of the temporal bone has been the making of clay models of the bone, especially clay models of the labyrinth. He thought he had a pretty fair knowledge of the gross anatomy of the labyrinth until he undertook to make clay models from Wood's model cast, and he found he knew very little about it until after he had made a number of models, and he is constantly finding things as he has occasion to work with the clay that had previously escaped him. He thinks that it is an excellent plan to make models of different parts of the middle ear, and the internal as well. He has noticed a great variation in the attachment of the tendon of the stapedius muscle to the stapes. Most of the textbooks say that it is attached to the head of the stapes. He has found it attached to the long process of the incus, but more frequently to the neck of the stapes or its posterior crus, than to the head of the stapes. He thinks much is to be accomplished by careful study; indeed, it is absolutely essential, if we are going to do efficient ear work, that we put a great deal of time on the anatomy of the ear.

Dr. George W. Boot has been under a different impression than Dr. Cavanaugh, for in his specimens of temporal bones at birth the annulus is not separate from the squama.

With reference to dehiscences of the floor of the tympanum, large dehiscences which would permit the jugular bulb to be seen through the membrana tympani are certainly not very frequent. In ten years' work at the Central Free Dispensary he had seen but one case in which the diagnosis had been made.

One point about the anatomy of the tympanic cavity was not mentioned by Dr. Cavanaugh, and this is the distance from the manubrium to the promontory. Most of the textbooks are erroneous in that they represent this distance much greater than it should be.

The cellular spaces mentioned by Dr. Cavanaugh as extending from the upper part of the tympanum into the upper wall of the canal are of considerable importance, for the burrowing

of pus into these cells occasionally causes the formation of a fistulous opening into the canal some distance external to the membrana tympani.

Dr. Cavanaugh gave the thickness of the drum membrane as about 1/10 mm. It is remarkable to what extent this thin drum membrane can swell in suppurating conditions of the middle ear. It is not uncommon to see a drum membrane which is 2 or 3 mm. in thickness as a result of suppuration.

He hoped Dr. Cavanaugh would pardon him if he called his attention to two errors in nomenclature. The manubrium is not the long process of the malleus, and the antrum is not the mastoid antrum, but is the antrum tympanicum.

Regarding the supposed change in the position of the drum membrane, it seems to him this question could be quite easily settled by measuring the angle which the groove of the annulus tympanicus on one side forms with the groove of the annulus tympanicus on the other side in the newborn and in later life.

In this connection, he called attention to two or three specimens on the table, one showing an anomalous course of the canal of the chorda tympani. Another specimen showed the relation of the antrum to the tympanum at birth, at which time the aditus is almost directly above the tympanic cavity.

Dr. Joseph C. Beck was sorry that the essayist had skipped a large part of the paper. The reading of the same will be of great interest; also the illustrations.

Regarding dehiscence, he called attention to the work of McKernon in infants, particularly in the region of the jugular bulbs, which are very numerous, contrary to what Dr. Boot said.

Regarding the cellular spaces referred to, you will find in X-ray pictures cellular areas reaching from the tympanic cavity to the sphenoid as one continuation of cells. You must deetrmine whether these socalled cellular spaces are really cellular spaces in connection with the cavity lined with this epithelial mucous membrane, or are of diploeic bone. He has some histologic sections of this particular portion, and has found many of them are diploe.

Dr. Cavanaugh, in closing, wished to thank the members for discussing his paper. Owing to the lateness of the hour, he omitted reading the entire article, which would have cov-

ered the remarks of Drs. Shambaugh, Boot, Andrews and Beck, but he mentioned that when the article appeared in full it would be found to deal with their suggestions.

Regarding Dr. Boot's statement that a dehiscence is rare in the floor of the middle ear, Dr. Cavanaugh wished to say that he has seen that condition in a pathologic specimen.

As to the folds of the tympanic cavity, discussed by Dr. Shambaugh, reference was made to them in the paper, but omitted for lack of time.

Paper: International Otological Congress in Boston, 1912: (a) Otosclerosis; (b) Discussion of the Demonstrations of Professors Urban Pritchard and Denker; (c) Heath's Paper.

Dr. J. Holinger said that the demonstration of microscopic specimens of Professor M. Pritchard, of London, and Professor Denker, of Halle, contradicted each other. Professor Pritchard said that birds have no basilar membrane in the labyrinth; Professor Denker showed very beautiful specimens of it. This finding is important for the theories of hearing. The same is true of specimens of Professor Siebenmann, who showed labyrinths of guinea pigs where the incus was removed from one middle ear, and the animal exposed to noises calculated to produce degeneration of Corti's organ. The cochlea where the incus was removed remained normal, the other one was degenerated. Both of these findings speak in favor of Helmholtz's theory of hearing, and against Zimmermann's.

Finally, attention was drawn to a demonstration of Professor Siebenmann of specimens and drawings of otosclerosis. The pathology of this disease was attacked in a book by Professor Manasse, and also the diagnosis as given by Bezold. The specimens counter-prove Professor Manasse's attacks.

DISCUSSION.

Dr. J. Gordon Wilson said that he read Manasse differently from Dr. Holinger. He understood Manasse to say that otosclerosis is primarily a hereditary degeneration. If he remembered Manasse's pictures aright, they showed in the labyrinth capsule as the earliest sign a collection of what he called granulation tissue in close relation to an artery. Then

followed absorption of the old bone and redeposit of new bone. Manasse did not believe it to be a specific disease of the stapediovestibular attachment, but of the labyrinth capsule. Subsequent to this come the changes in the nerves of the cochlea and the cells of ganglion spirale.

Of course, the whole question is still extremely obscure, but it is satisfactory to find two opposing views so energetically discussed. The outcome cannot but give a more accurate knowledge of the etiology and pathology of this obscure disease.

Dr. George W. Boot said, with reference to the presence or absence of a basilar membrane in birds, that he had cut sections from the lagena of the owl, bluejay, woodpecker and the common barnyard fowl, and that in these birds at least a basilar membrane was present.

Dr. Shambaugh called attention to one point which the specimens exhibited by Dr. Urban Pritchard and Dr. Denker brought out. Dr. Urban Pritchard has held the view that the cochlea in the bird's ear has but two scalæ—a scala tympani and a scala media—and that there is no Reissner's membrane. Dr. Denker holds the view that the scala vestibuli is present. The Reissner's membrane is quite a different structure in birds from what it is in mammals, but it is still there. Dr. Urban Pritchard showed specimens of the labyrinth taken from the ornithorhynchus, the duck-bill of Australia, which showed that the cochlea in this interesting animal possesses the characteristics of the bird's cochlea rather than those of the mammal.

Dr. Joseph C. Beck said that a great many of the cases operated on by Mr. Heath in Boston had required reoperation. Mr. Heath is an enthusiast and has created a semiradical operation and given some people a moment to think before operating every ear radically. So far as the speaker was concerned, he has not been very successful in the use of Heath's operation, except in children, and he thinks that the indications for the operation are rather the point that Mr. Heath leaves out, and the pathologic conditions that are found in the bone.

Dr. Beck is interested in the pathology of otosclerosis, and especially in the work of Gerber. Gerber, in his opinion, is the coming man. His differentiations coincide with those of

Gray in the study of otosclerosis, and also with the processes that are found in osteomalacia and rickets, and in some forms of degeneration as they are found in asthmatics in the upper respiratory tract. The speaker has been working along this line, and has had a good deal of correspondence with Dr. Gray in reference to this, and it is, in his opinion, a subject of great interest.

He believes that the standpoint of Manasse is worth while considering.

Dr. Holinger, in closing, said that the standpoint of Manasse certainly is worth while considering, otherwise we would ignore it. Every criticism has to be taken into account, but he thinks it is necessary to weigh the merits and demerits on both sides. The idea of spongifying being an inflammatory process leaves us absolutely in the air as to the clinical course. The word "inflammation" tries to explain the pathology, but no reason is given why it is more justified than "abnormal growth."

With reference to the question of inflammation: We have repeatedly heard it said that the identification of pathologic processes of the bone is one of the most difficult problems. Dr. Holinger has studied the work of Manasse quite recently, and has seen the areas described by Dr. Wilson, which are circumscribed with lines in the picture. But Professor Manasse does not say why he calls these areas inflammatory, or why they might not be due to abnormal growths.

Dr. Holinger states that Siebenmann has examined a great many young individuals, while Manasse has no patient below forty. Later on there are new formations, and just that circumscribed focus in the very old individual explains it.

Dr. Wilson said he did not think so.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of March 18, 1913.

DR. J. GORDON WILSON, PRESIDENT, IN THE CHAIR.

Stenosis of Right Auditory Meatus.

DR. C. H. LONG reported the case of Miss J. C., aged twenty-one years; lives at home. On September 13, 1912, she presented herself with pain and a discharge of the right ear. Family history negative. Personal history: Always healthy until the age of nine years, when she had the measles. From that time until her eighteenth year (1910), she suffered from phlyctenular keratitis, the corneal opacities becoming so opaque that her vision in both eyes was often as low as 15/200, even with the help of her correcting lenses.

The advice for the clearing out of the throat was not followed until April, 1912, when the phlyctenules reappeared.

In April, 1910, when the eye trouble had abated, an acute eczema appeared around the alæ, bridge of the nose, and side of the cheek. About September 5, 1912, an itching, burning sensation was felt in the right ear, followed by pain and a watery discharge. On the 13th he examined the ear, and, in view of her past history, was unable to make an exact diagnosis. The microscope showed only the presence of pneumococci. September 24th, the symptoms demonstrating a rather deep infection, with the possibility of pus formation, he made a free incision through the most sensitive part to the bone. This was done under gas anesthesia. No pus was found, but immediate relief from pain ensued. Cleansing with mild antiseptic solutions followed, but without much, if any, benefit. A thin, mucopurulent discharge came from the inner side of the narrowed opening of the external auditory meatus. It was impossible to view the drum membrane, so there was no way of telling the condition of the middle ear. The hearing was lowered, to be sure, but no more than we should expect with an occluded meatus. The lumen continued to become smaller, so that by January, 1913, three months from the be-

ginning of the infection, the meatus scarcely allowed the passage of a small probe. He tried stretching with graduated sounds, then inserting different varieties of drain, sterile tin foil, moulded in the shape of a tube, rubber tubing, and gauze, but with unsuccessful results. He then used a slippery elm tent, cutting it down to fit the hole, taking care that the string was strong and properly tied, so there would be no danger of the swollen wood slipping behind the stricture. A second and larger tent followed the first. As a result of the dilatation, the drum membrane was inspected and found intact. In two or three days the meatus was nearly as small as before. It was at this stage that he presented her to this society.

On January 21st, under ether anesthesia, he performed the Körner flap operation. In a couple of weeks nearly all discharge had ceased and all wounds healed, with the exception of a spot external to the stricture in the meatus, where the flap did not cover the cut surface. This is gradually filling in, and he hopes soon to report a successful termination of a seemingly simple infection, but one that baffled all ordinary therapeutic measures. In this case he had to deal with an acquired occlusion of the canal, due to an infection seemingly deeper than the glands and hair follicles. Vaccines were not used in this case, owing to previous failures in former furuncular cases.

Referring to the flap operation, he should have met with greater success had he inserted a graft on the raw surface or else used the Jansen modification of Körner's operation.

Otosclerosis.

Dr. J. Holinger presented a series of specimens of otosclerosis presented to him by Prof. Siebenmann, of which some had been shown before. He had one new one which had two foci, one of which does not reach the surface at any place. From this it may be argued that under certain circumstances the process may start in the intermediary part of the bone, without touching the surface of the periosteum. This is an argument against the inflammatory nature of the process. He also showed one specimen of artificial boiler maker's deafness in a guinea pig, in which the incus was removed in one ear. Corti's organ in the operated ear was intact; in the non-operated one it was degenerated. Proof for Helmholtz's the-

ory. He also showed one specimen of the labyrinth of a guinea pig which was exposed for fifty nights to a whistle with the clear sound of small "g." It shows circumscribed degeneration of Corti's organ.

Gross Lesions of the Middle Ear.

Dr. J. Gordon Wilson presented specimens showing gross lesions of the middle ear. They were obtained from patients in a lunatic asylum who showed marked hallucinations of hearing. It is interesting to note that in cases of aural hallucinations there are found a large percentage with marked ear disease.

Acute Mastoid Abscess and Temporosphenoidal Abscess.

Dr. Wilson also showed a young lad of twenty-one on whom he had operated three weeks ago for acute mastoid abscess and temporosphenoidal abscess on right side. The lad had before the operation severe pain in the right parietal region, which dated back to an accident four weeks before Dr. Wilson saw him. The X-ray pictures showed two fractures of the skull, one in the parietal and another in the squamous temporal. A full history will be published later.

Esophageal Bougie Fitted With a Wax Spindle-Shaped Enlargement for Dilatation of Cicatricial Strictures of the Esophagus.

Dr. Otto T. Freer showed an esophageal bougie fitted with a wax spindle-shaped enlargement for dilatation of cicatricial strictures of the esophagus. Dr. Freer was led to create the device by a case of stricture of the esophagus just above the cardia, which he had been keeping open for some years by monthly passage of the usual ivory olives, an especially made hard rubber olive, seven-eighths of an inch in diameter, completing the dilatation. Of late, however, the esophagus had begun to pouch above the stricture, so that the olives merely pushed down this pouch and failed to pass. A flexible gum elastic bougie, No. 20, however, entered the stricture readily enough. In order to accomplish dilatation with this bougie, Dr. Freer wound a spindle of dental floss silk upon it at the junction of its lower and middle third, and made the spindle solid by painting it with boiling hot dental moulding wax. The surface of the spindle was finally smoothed with a layer of

still fluid but cooler wax, and the form of the spindle was finally shaped by scraping it with the edge of a knife. The upper end of the bougie was then cut off and a whalebone rod introduced as far as the spindle to stiffen the upper portion of the bougie. The spindle readily followed the flexible distal third of the bougie through the stricture and dilated it easily.

Dilating spindles of the kind described may be made of any desired size upon the regulation bougie. Dr. Freer regards these dilators as superior to distensible rubber bags, which are harder to pass than the ordinary olive.

DISCUSSION.

Dr. Joseph C. Beck said that Dr. Wilson's case had been exceedingly interesting to him. Regarding this socalled dead area—or the area in which diagnosis is very difficult—he had had a personal experience recently. His brother had succumbed to a tumor located in this region. That a tumor the size of a small apple can exist in this area without producing any symptoms is certainly a strange thing to him, and he is studying his brother's case from every point. This subject is of extreme interest to all rhinologists and otologists, because he believes it is in their sphere to assist in making the diagnosis. It is surprising that such an amount of pus could be obtained in this area without causing any symptoms other than pain.

Dr. Shambaugh referred to a case of brain abscess which had recently come under his observation. The chief complaint of the patient was severe onesided frontal pain of several weeks' duration. When first seen by Dr. Shambaugh there was a subnormal temperature, slow pulse and complaint of unbearable pain over the left eye. The patient stated that he had lost ten pounds in weight, and the clinical condition suggested something decidedly more severe than a frontal sinus abscess. Examination found the septum deflected to the left, which made it impossible to catheterize the frontal sinus. A mucopurulent secretion was found oozing from the middle meatus. Under cocain the anterior half of the middle turbinal was easily removed, and by cauterizing the discharge was found to come from the frontal sinus. The patient seemed decidedly better for a short time after the removal of the middle turbinal, but very shortly the frontal pain returned.

It was decided that there was probably a brain abscess. Several neurologists examined the case and agreed with the diagnosis, but were unable to locate the abscess. There was no middle ear trouble, and since the frontal sinus was infected, it seemed probable that the frontal sinus had some relation to this infection. After opening the frontal sinus the condition found did not indicate a process which was likely to invade the brain cavity. There was no perforation of the posterior wall of the sinus. However, it was thought best to remove the posterior wall in order to make sure that there was not an extradural abscess in this region. None was found. Still believing that there was a brain abscess and probably located in the frontal lobe, an opening was made higher up and the bone trephined. No abscess was found. After about a week symptoms of right sided paresis appeared, which pointed toward a probable temporosphenoidal abscess. The bone was trephined and a large abscess was found in this region. The colon bacillus seemed to be the active organism.

An interesting feature of the case was that the patient showed marked aphasia for names of objects; while recognizing the object itself, he could not name it. The patient was left handed, and the abscess was located on the left side, but the location of the speech center in his case was on the affected side—the left side—because he had been taught to do one thing with his right hand, and that was to write.

Dr. Otto T. Freer did not think that Dr. Shambaugh was clear in the pathology in the case he reported, and he especially failed to show a connection between the frontal sinus opened and the abscess found. While there are cases where, by way of lymphatic infection through bone, brain abscess has been produced, these cases are in the nature of curiosities, and brain abscess due to frontal sinus suppuration practically always implies ulceration of the mucous membrane of the posterior wall of the sinus, caries of the bone and infection of the dura.

Dr. Freer did not regard the signs which led Dr. Shambaugh to a diagnosis of frontal sinus suppuration as enough to warrant such a diagnosis. Pus in the middle meatus may mean suppuration of the antrum of Highmore or anterior ethmoidal cells, instead of suppuration of the frontal sinus, for the pus from all these cavities appears in the middle meatus. Pain over the frontal sinus is a very uncertain sign of

frontal sinusitis, for it is often due to supraorbital neuralgia, and even oftener, in Dr. Freer's experience, is reflected pain due to suppuration of the maxillary antrum, with a healthy frontal sinus underneath the region of the pain. Dr. Freer has often seen rhinologists misled by brow pain into a diagnosis of frontal sinus suppuration, when the disease was a suppuration of the maxillary antrum. Dr. Charles E. Parker, of the Golden Square Hospital, London, and Dr. Brown Kelly, of Glasgow, informed Dr. Freer that pain over the frontal sinus, in their experience, was very often caused by maxillary sinusitis with a healthy frontal sinus.

Dr. J. R. Fletcher had seen a large abscess of the frontal lobe emanating from frontal sinus empyema on the right side, but the relatives would not allow an autopsy.

Dr. Clark W. Hawley reported a case that he operated on last summer. An extensive mastoid dissection was done and quite a portion of the middle fossa and the lateral sinus was uncovered. The convalescence was very slow at first, but he began to show all the signs of brain abscess, temperature down to 94°, pulse to 54, weakness and other symptoms. Several consultations were had. It was decided that the indications were not sufficiently well marked to justify opening the skull, and the result proved the wisdom of it. From this relapse he seemed to be recovering, when he suddenly died one night. A postmortem was done, and the most careful dissection of the brain showed no abnormal change; no abscess or meningitis could be demonstrated. This shows that there are two sides to the subject, and that we cannot always depend on lowered pulse rate and temperature as indicative of brain abscess.

Double Fistula (Right and Left), the Right Being Probably in the Horizontal Semicircular Canal, the Left in the Oval Window.

Dr. J. R. Fletcher demonstrated two cases, as follows: First, that of a man, about forty-five years of age, with a double fistula (right and left), the right being probably in the horizontal semicircular canal, the left in the oval window. The drumheads were both adherent to the labyrinth wall. The fistula left was plainly visible. The man had complained of vertigo when blowing his nose. This symptom caused him to consult Dr. Fletcher. Knowledge of the fistula symptom

directs one's attention immediately to the ear, although this man did not complain of his ear at all. Examination revealed cholesteatoma in the external auditory canals, both right and left. The interesting feature about the right fistula was that in doing the Valsalva he developed pronounced vertigo, with true vestibular nystagmus toward the diseased side. Inspection of the drumhead at the time of the blowing showed a distinct track which the air followed. This was located along the inferior margin of the drum. No change took place in the left drumhead. Both sides responded to the fistula test, each causing nystagmus to the right. There is as yet no satisfactory explanation why the fistula test in one case makes a nystagmus in a certain direction, and in another case in the opposite direction; in other words, we cannot explain the lack of constancy in the direction of the flow of the endolymph in the canal under seemingly like circumstances. Before the removal of the cholesteatomata there was no response to the fistula test. It seems to Dr. Fletcher that this case shows clearly the value of the fistula symptom.

The second case was one of complete

Destruction of the Function of the Right Labyrinth Following a Radical Mastoid Operation.

There is a fistula present in the lateral arm of the right horizontal semicircular canal, which can be easily seen. The excretion from this fistula is serum. This may be a case of serous labyrinthitis following the radical mastoid. As Dr. Fletcher did not do the operation, and the history previous to the operation is not clear, positive statement cannot be made. The case is interesting because the patient responds very faintly to the turning and the caloric tests of the good ear, much more slightly to the turning test, and not at all to the caloric test of the bad ear. This case illustrates the readjustment which takes place after the functional destruction of one labyrinth, namely, the assumption of the function of both labyrinths by the single good one. The use of the Bárány noise apparatus for the exclusion of the good ear was also demonstrated in this case. During its use the patient was totally unable to hear very loud spoken language.

Paper: Clinical Vestibular Nystagmus.

By John R. Fletcher, M. D.

Dr. Fletcher said it is quite important, in familiarizing ourselves with the use of vestibular nystagmus in otology, to remember that there are many cases which do not come under the rules, and that all cases of this type of nystagmus do not originate in the ears—that is, they are extracranial, as, for instance, the nystagmus with vertigo from auto- and other intoxication, uterine vertigo, etc. No direct ear symptoms and none pointing directly to the cranial contents will speedily put us right on these cases. Other cases, manifestly of the gravest, are at present quite beyond diagnostic certainty and are helped but little by the symptom under consideration. However, in diagnosing and differentiating serous and suppurative, circumscribed and diffuse, manifest and latent labyrinthitis; hyperemia and anemia of and hemorrhage within the labyrinth; and retrolabyrinthine disease from one another and from nonotologic diseases, its place is well fixed and its value great. The patients we see are referred or come of their own accord; we are asked to see the first because of the nystagmus, and are consulted by the second because they have vertigo. They know nothing of the nystagmus, but for various reasons believe their vertigo comes from the ear or nose; as, for instance, the first patient demonstrated by him that evening, who came complaining of brief attacks of severe vertigo, with sometimes nausea, whenever he blew his nose. Careful examination determined beyond doubt that he had a fistula in both ears, which is uncommon, and, further, that but one of them is affected by the nose blowing. We learn the latter from the fact that performing the fistula test on the left side produces nystagmus in the same direction as by blowing the nose, but its velocity and excursion are not so great. If we make the same test on the right, the result is identical with the nose blowing. This and the unique fact that the route of the air current from the eustachian tube can be followed with the eye make it certain. In addition, a pinhole perforation of the closely adherent left drumhead, directly over the oval window, designates the site of the fistula. Moreover, this per-

foration would allow air coming from the tube to escape. Another point to make is that gentle pressure over the site of the perforation with a probe wound with cotton causes nystagmus, much the same as the fistula test. The latter is made by carefully blowing air into the external auditory canal through a hollow olive-shaped hard rubber point from a small rubber bag and tube. The fact that his hearing is good beyond expectation signifies that the disease is circumscribed. Air can be prevented from escaping through the eustachian tube when it is wide open and the drum membrane is perforated, by the patient holding his nose and blowing at the time the test is made, as suggested by Ruttin, or by closing the tube with cotton within the middle ear, when possible.

Of the three clinical tests, the fistula test, turning, and the caloric test, the latter is the most constantly useful. The speaker's second patient brings out this point quite well. Her response to the fistula test is negative, though one is visible on the right, and freely exposed to the pressure of air because of tubal closure after the radical mastoid operation. She is totally deaf in this side. Schwabach localized in the left; Rinné negative +. She responds very slightly to turning, either to right or left—so slightly that unless very closely observed one might report the response absent. Her response to the caloric test on the right is negative. All these facts signify beyond a doubt total functional inactivity—a functional death of the right labyrinth. The whole exposed lateral labyrinth wall was irrigated with ice water for a long time without result. The same water used in the left ear caused horizontal and rotatory nystagmus to the diseased side, but not so promptly nor so vigorously as in the normal person. Quite warm water caused to flow into the left ear caused a rather feeble rotatory nystagmus to the good or irrigated ear. These caloric tests to the left ear tell us that her left labyrinth is functionating. If we had no other information than the caloric test given us, we could safely diagnose a dead labyrinth on the right and a functionating one on the left, though the left response is normal only in the direction and plane of the nystagmus, not in quickness and degree. In other words, what is known as readjustment has taken place —the good labyrinth is somewhat less keenly doing the work of both. Their action and reaction on each other has been

lost—there is no longer synchronysm. The normal reaction to turning a patient ten times around the vertical axis is nystagmus in the direction of turning during turning, and in the opposite direction after turning. In turning this patient to the right there is no after-nystagmus, and the usual vertigo is absent. In turning to the left the after-nystagmus is so slight as to amount almost to nothing. When we use the turning test to discover pathologic labyrinthine conditions, the rule is that if one labyrinth gives an after-nystagmus of at most half the duration of the other, the labyrinth giving the short-enduring reaction is pathologic. In this case one does not respond at all and the other barely. We may interpret this as nothing, equaling the half or less of almost nothing; that is to say, within the rule. Turning must always be used in case of dry perforations, as irrigation is not to be thought of on account of the danger of causing acute suppuration. Of course, the caloric test may be made with clean air, warm or cold, or any other cleanly method which may be devised to change the temperature of the labyrinth wall, except the use of fluids, as above. The turning tests of Dr. Fletcher's first patient have not been made, as he objects strongly to the vertigo.

Let us suppose a patient complains of vertigo coming on in attacks of from a few seconds to a few hours' duration, of a light or relatively severe grade, with the history of an old chronic suppurative otitis media. We must examine for spontaneous nystagmus by directing the patient to look to the extreme right and left without turning the head. If in this eye position we find nystagmus directed to one or both sides, we may regard the case as circumscribed labyrinthitis; but to make the diagnosis more certain the caloric reaction should be positive, with change of direction of the spontaneous nystagmus to the use of cold when to the right, and of warm water when to the left. There should be a response to the turning test, with the comparative difference mentioned above. The hearing test should be positive. The fistula test should be made, and will usually be found positive; though a fistula may be present without a positive response to the test, due either to the escape of the air or to some obstruction to pressure, such as cholesteatoma, granulations, etc. Diffuse serous labyrinthitis occurs in acute suppurative otitis, chronic sup-

purative otitis media, and follows the radical mastoid operation. The characteristic nystagmus is toward the sound side, rotatory and severe. Not infrequently the circumscribed changes to diffuse serous or diffuse suppurative labyrinthitis. The question of a labyrinth operation is thrust before us, and is settled by the differential diagnosis. In both cases the nystagmus will be directed to the sound side and is severe. If any sign of functional activity of the diseased side remains, namely, response to the caloric, turning, fistula and hearing tests, the case is one of serous labyrinthitis. Diffuse suppurative labyrinthitis is totally destructive and very dangerous to the cranial contents. In such case there is no response to tests, and so far as they are concerned we must diagnose diffuse suppurative labyrinthitis. Unfortunately, we have reports of operated cases in which all reactions failed which were not suppurative, and others in which the labyrinth operation was done which recovered. We must, therefore, conclude that the serous labyrinthitis sometimes causes the picture of diffuse suppurative. These cases cannot be clinically differentiated from the diffuse suppurative. There is a great deal more to be said about serous labyrinthitis, but as we are dealing more particularly with the symptom—nystagmus—from the clinical standpoint, it may be well not to go too far afield, but proceed to the next and most dangerous disease of the labyrinth, namely, diffuse suppurative labyrinthitis.

No picture is quite the same as that of the serous. Severe vertigo, nystagmus to the sound side, nausea, vomiting, and disturbance of equilibrium. This is the manifest type. Latent diffuse suppurative labyrinthitis progresses more slowly. The history will show complaint of attacks of vertigo at intervals of greater or less time through the course of years. There will be no response to any of the function tests of the labyrinth —hearing test negative, caloric test negative, turning test negative, and no fistula symptoms. The only possible symptoms are, first, very small spontaneous rotatory nystagmus, both right and left, when the glance is in these respective extreme directions. Second, the marked difference in the duration of the after-nystagmus following turning to the right and left, head erect. Ruttin gives the difference as from one to three to six. Let us suppose the right labyrinth to be the seat of the trouble—so long as true compensation, as defined

by Ruttin (Klinik der Serösen und eitrigen Labyrinth-Entzündungen), has not taken place, the result of turning ten times to the right, head erect, will be an after-nystagmus to the left of from fifteen to thirty seconds' duration, while turning to the left, used in like conditions, created an after-nystagmus of about five seconds' duration. When complete compensation has taken place, that is, when not the least particle of function remains in the diseased labyrinth, the sound side assumes the function of both sides, but somewhat less acutely, as follows: The after-nystagmus following ten turnings, either to the right or left, head erect, in the opposite direction to turning for ten to fifteen seconds. In other words, the after-nystagmus again becomes equal to both sides, differing from the normal in that its duration is reduced to one-half or one-third or less. The sound labyrinth does all of the work, but is only able to produce half the result in point of duration. Several authors have reported necrosis with sequestration of a part or the whole of the labyrinth. These cases, of course, are latent suppurative labyrinthitis, and sequestration may be considered the last symptom, unless we add facial paralysis, which now takes place. Ruttin mentions two cases in the book above named in which the fistula symptom was present after sequestration, showing that some filaments of the nervous end organ yet remained. The early picture seen in traumatic destruction or hemorrhage into one labyrinth is quite the same as in diffuse suppurative and extreme serous labyrinthitis, but here we are not dealing with a case of old suppurative otitis media, while circumscribed serous and suppurative labyrinthitis of necessity follows otitis media. On the other hand, it must not be forgotten that an otitis media may follow the trauma. The trauma caused by the radical mastoid operation not infrequently converts a circumscribed into a diffuse serous labyrinthitis. The nystagmus characterizing (though we rarely see it) the circumscribed labyrinthitis should not change after the radical operation. If it does, it is spontaneous rotatory and directed to the sound side. The great question, then, to be decided is between the serous and suppurative diffuse labyrinthitis. Clinically, today we must consider all cases showing no reaction to the fistula symptom, hearing, caloric and turning tests, with strong nystagmus to the sound side following chronic sup-

purative otitis media, diffuse suppurative labyrinthitis. The accompanying symptoms of nausea, vomiting and disturbances of equilibrium are, of course, usually present.

While the above relating to nystagmus is the rule, in a considerable number of cases of suppurative labyrinth destruction there is a horizontal nystagmus to the diseased side. Further, the nystagmus to the sound side may be compound; that is, both horizontal and rotatory. When there is a nystagmus to both sides by glancing in the extreme lateral direction, one may be confused who thinks of the rule that nystagmus is increased when the patient glances toward the quick component and decreases or stops completely when the glance is in the opposite direction. An important fact is that the nystagmus emanating from the labyrinth is strongest at the beginning, gradually growing weaker and weaker until it ceases completely.

It is difficult to write of clinical nystagmus and keep strictly to the text when it comes to the consideration of serous and suppurative labyrinthitis, because the nystagmus is the same in both cases during the time when the differential diagnosis must be made, if possible, though the attempt has been made to direct attention to the nystagmus throughout the paper. The real danger to the life of the patient is through intracranial, that is, extralabyrinthine, complications, of which there are two in particular, from labyrinthitis, abscess and meningitis. Fortunately the nystagmus serves us here, and that, too, in the midst of the symptom storm.

In cerebellar abscess the nystagmus may be directed to the sound as well as to the diseased side. Characteristic for this nystagmus is it that as the disease progresses the nystagmus becomes more intense, ultimately reaching such a grade as is never seen in labyrinth disease.* In labyrinth disease the nystagmus to the sound side predominates and, last, after that which may have been directed to the diseased side ceases. If this nystagmus suddenly changes to the sound side and is strong, with tendency to increase rather than diminish, cerebellar abscess may be diagnosed confidently. Other cases may be doubtful, but this may be said with certainty, that if the complete labyrinth operation has been done and nystagmus

*H. Neumann: Zur Differential Diagnose von Kleinhirnabszess und Labyrintheiterung.

persist to the diseased side, there is an intracranial complication. (Physiologie und Pathologie (Functions-Prefüng) des Boyengang-Apparates beim Menschen.) Bárány says: "Is the right vestibular apparatus unirritable, with a strong rotatory nystagmus to the right, so is the same caused by an intracranial disease." A dead labyrinth cannot originate a nystagmus toward its own side; therefore, the irritation must come from somewhere along the course of the vestibular nerve or its center.

The differential diagnosis between cerebellar abscess and meningitis of the posterior fossa cannot be made by means of the nystagmus. It is, in fact, usually difficult, though the other symptoms are the real guide and frequently do not fail us.

To those who wish to follow the subject more deeply, Neumann's "Der Otitische Kleinhirnabszess" will be of great value.

DISCUSSION.

Dr. William L. Ballenger said the average otologist was very much confused by the theoretical problems presented in a study of this question. As a matter of fact, there is one class of cases at least that can be diagnosed without knowing anything about nystagmus, as, for example, latent diffuse suppurative labyrinthitis, with total deafness and a history of a vertiginous attack at one time in the past—it may be weeks, months or years previously—and nystagmus might have been observed at that time. Since that time there has been total deafness and a discharging ear. Those cases are referred to as diffuse suppurative latent labyrinthitis. One might be able to make a diagnosis of a case of this sort, even if he has not made a study of the deeper problems presented by Dr. Fletcher. It is easy for an otologist to know that he has a case of total destruction of the labyrinth. If we have, then, a case with a chronic discharge and the patient more or less deaf, the noise apparatus, the stream of water, or the tuning fork, as suggested by Dr. Pierce, inserted into the meatus, we can determine whether a patient has total destruction or not, and then, with a previous history of a vertiginous attack, we can arrive at a fair estimate that we have a case of diffuse suppurative latent labyrinthitis. So there is one type of case that may be fairly

well diagnosed without the classical tests. Dr. Ballenger referred to a case under his observation at the present time of that type, which had been operated on ten or twelve years ago —three times within a period of a year or two—for mastoid disease. This man did not know he was totally deaf in the operated ear, and the speaker did not know it until he had been around the office for two years. He had been treating him for frontal sinus disease, and had done an operation on him for it. One day he complained of dizziness, which aroused Dr. Ballenger's suspicions. He used the noise apparatus, and found him totally deaf in the operated ear.

So the problem is not so complicated as at first thought. It is true that these more theoretical aspects of the subject must be mastered before we have complete mastery of the subject, but it has been his desire to approach the subject from the simpler standpoint. He has seen nine labyrinth cases during the past year—most of them of the latent type, with a history of vertiginous attacks eight or ten years previously. Ruttin says that when the labyrinth has been long destroyed we may get complete compensation; that is, the nystagmus after the turning test is equal in either direction, though of shorter duration than in normal cases.

Dr. Norval H. Pierce said that Dr. Fletcher had presented the known facts that can be used in clinical work very well. He did not think he had anything to add. He carries out the examinations about as Dr. Fletcher does.

Dr. Robert Sonnenschein asked whether a Wassermann had been made in the case of the woman who had lost the reaction to turning, but still had the caloric. At a meeting of the Vienna Otological Society, held about two years ago, Neumann stated that the fact of loss of turning reaction and retention of caloric reaction almost always suggests lues.

Dr. Fletcher said the woman had had an abdominal trouble for which she was operated. A Wassermann was made and was negative.

Dr. George E. Shambaugh stated that there is usually no difficulty in recognizing an involvement of the labyrinth in connection with suppurative middle ear disease. If the labyrinth involvement develops suddenly, there are symptoms of disturbed equilibrium, including nystagmus, which are always present. If the labyrinth involvement is one of long stand-

ing, especially where there has been a destructive process, the symptoms of disturbed equilibrium may be largely absent, but in these cases the labyrinth involvement can be diagnosed by the fact that the function of the labyrinth on that side is destroyed. In these cases of labyrinth involvement there may be some confusion in deciding on the proper treatment. Dr. Shambaugh thinks that the most important thing clinically is to determine when an operation on the labyrinth is indicated and when it is not. In all cases of acute labyrinthitis, where the disease stops short of a complete destruction of the function of the labyrinth, all are at present agreed that no operation upon the labyrinth is called for. These cases are usually classified as cases of serous labyrinthitis, and unless they develop into diffuse suppuration of the labyrinth, the development of an intracranial complication is extremely unlikely. It is only in cases of diffuse suppuration of the labyrinth, where there is already a complete destruction of the labyrinth function, that the question of a labyrinth operation is at all considered. In view of the large number of cases which recover spontaneously after a diffuse suppuration of the labyrinth, the question naturally arises whether we are able to make a distinction clinically between the cases of diffuse suppuration of the labyrinth which call for a labyrinth operation and those in which a labyrinth operation may not be performed.

In examining cases where there has been a long standing purulent otitis media, one frequently discovers a totally dead labyrinth. It is not necessary to subject all of these cases to a labyrinth operation. It seems probable that only those cases of chronic purulent otitis media, where the indications exist for a radical mastoid operation, that is, where there is an invasion of the temporal bone, with or without cholesteatoma formation, that the labyrinth operation should be performed. The question may not be so simple in cases where one observes in purulent otitis media the development of an acute labyrinth suppuration. There are some who believe that in all of these cases the labyrinth operation should be immediately performed. The speaker has not been able to subscribe completely to this opinion. He referred to a case that he had observed over a year ago, who had what appeared clinically to be a mild acute otitis media with no severe pain and very slight rise in tem-

perature. The patient recovered from a sudden destructive labyrinth involvement without an operation, either upon the mastoid or the labyrinth. Testing the ear one year later showed that there had been no return of function. In a similar way he had recently observed in a case of chronic purulent otitis media the sudden incomplete destruction of the vestibular part of the labyrinth. This case was not operated. The type of the middle ear suppuration clearly indicated a process which had not invaded the temporal bone. There was no evidence of caries or cholesteatoma. There was a large, central perforation and a history of recurring acute exacerbation of middle ear inflammation running over a number of years. The patient made a good recovery, but there was no return of function to the semicircular canals. The speaker believes that in many cases of acute labyrinth destruction from suppuration one may defer an operation upon the labyrinth, awaiting the development of the first symptoms indicating an intracranial extension. Of course, in cases where the clinical conditions already indicate the necessity for a mastoid operation, whether these symptoms occur in a case of chronic or in a case of acute otitis media, the mastoid operation should be performed, and at the same time the operation upon the labyrinth should be done.

DR. BALLENGER did not think it was right to let Dr. Shambaugh's statement stand. He operated on a mastoid case several years ago and did a radical mastoid. The man had total deafness at that time—had diffuse suppurative labyrinthitis. The patient lived for five years in comparatively good health, but five years after the radical mastoid he died of meningitis. It is not safe to allow such cases to go without a radical labyrinth operation. Sooner or later meningitis or brain abscess is liable to occur. The labyrinth operation can do no harm, provided the operator knows the technic. We would not allow a chronic mastoiditis to go on indefinitely without operation, and there is infinitely more reason why we should not allow a labyrinth total destruction to go on indefinitely without operation, as intracranial complications are always liable to occur at any time. If operated, these cases are safe in that respect.

DR. SHAMBAUGH stated that in the case reported by Dr. Ballenger, where there was a long standing destruction of the labyrinth, no labyrinth operation was called for unless the indications existed for a radical mastoid.

DR. JOSEPH C. BECK said that the conservatism expressed by Dr. Shambaugh was certainly welcome from a man who knew so much about the question as Dr. Shambaugh. He had seen such a case as the one referred to by Dr. Shambaugh as cured that developed a very severe labyrinthitis with irritation of the dura, or meningeal irritation. He would certainly not operate on the labyrinth, but on the mastoid process.

Dr. Beck asked Dr. Fletcher whether, in the case of the man exhibited with two fistulæ, since this is an indication for operation, he would do bilateral radical operation in this case.

DR. SHAMBAUGH stated that in cases of acute otitis media where acute labyrinth symptoms developed, even when these symptoms indicated clearly a complete destruction of labyrinth function, he was not prepared to operate either upon the mastoid or upon the labyrinth unless indications were present for a mastoid operation, or unless the patient developed symptoms indicating an intracranial extension.

DR. NORVAL H. PIERCE said that it is his custom, in cases of acute or chronic otitis media, in which acute labyrinthine symptoms arise, to never operate on the mastoid for a simple mastoid or for a radical mastoid while those active symptoms were present, unless there are increasing meningeal symptoms. For proof of this he depends largely on lumbar puncture. He invariably allows these patients to remain in bed until all active symptoms have subsided, such as dizziness, vertigo, etc. In quite a series of cases he has not been disappointed in this method. Before adopting it, however, he was largely disappointed in the outcome of operations undertaken at the time of active labyrinthine irritation. He has seen fatal cases result from opening the labyrinth in the face of the active manifestations in the labyrinth, and he believes that we do not insure safety to the patient by simply opening the labyrinth. He believes that a given inflammation of sufficient virulence and clarity will proceed to the meningeal serous spaces, irrespective of whether we open the labyrinth or not. He therefore depends largely in these cases, while the labyrinth symptoms are in progress, on the chemical and cytologic examinations of the cerebrospinal fluid. In the absence of changes in the cerebrospinal fluid, the patient remains in bed at rest. They practically invariably recover from the active inflammation in the labyrinth, and then he proceeds with the radical operation, but very rarely feels called on to open the

labyrinth—very rarely. He thinks the terms serous labyrinthitis and suppurative labyrinthitis are misleading in a clinical sense. They have to do largely with the character of the inflammation, and you cannot always tell at the clinic whether it is a pure serous change or whether it is a suppurative change.

The point he wished to make is that he supports the position that we should not operate in the presence of a stormy, acute labyrinthitis, whether it is serous or not, whether there is a fistula or not, unless there are indications of advancing meningeal involvement. If there are such symptoms, he would open the labyrinth and drain the subdural spaces. In a chronic otitis media with a quiescent, destroyed labyrinth and a demonstrable fistula or sequestrum, he would open the labyrinth.

Dr. J. Gordon Wilson said that he was sure the society appreciated the review given by Dr. Fletcher of the clinical applications of nystagmus. The rules given by Dr. Fletcher are of very great use. But one must recognize their limitations and desire more definite information. This, he fears, we are not likely to obtain in the first place from man. Here the conscious element is ever coming into play, modifying and sometimes making negative our findings. It has always appeared to him that we must look to the physiologist for the fundamental rules governing nystagmus.

The side of nystagmus that has interested him particularly has been the experimental. Here one sees that the fundamental facts of labyrinthine nystagmus are relatively simple. In discussing nystagmus one difficulty constantly met with is that we fail to recognize that we are referring to two different things, a slow deviation which is labyrinthine, and the quick return which is extralabyrinthine. All our tests should have this in view. Thus he would rather that Dr. Fletcher spoke of the patient with labyrinthine disease falling in the direction of the slow movement of nystagmus. In man the falling in cerebellar lesions does not follow any such definite rule.

The careful observation of nystagmus in differential diagnosis is extremely important; for instance, in the differential diagnosis between cerebellar and labyrinthine disease. Here again the fundamental rules which will guide us must come from animal experimentation. For in man so many other influences may arise to complicate results. Thus in cerebellar

abscesses, for instance, we have absorption of toxins, pressure on the vestibular nerve, etc. Let us aim to get the fundamental rules; then we can learn to eliminate the unessentials.

Dr. Fletcher, in closing, referred to Dr. Ballenger's remark that "we can so easily, without these tests, diagnose a latent suppurative labyrinthitis," and later said that "he was very much astonished after a long time that the patient had vertigo." That means he never had acute labyrinthitis. He had serous labyrinthitis, because just exactly those cases recover, and the patients can again have symptoms of circumscribed labyrinthitis, such as attacks of vertigo.

Dr. Lewy asked about the action on acute suppurative otitis media in which there was a fistula present. The speaker was sure, from his own personal experience, that he could not answer this question, because had never seen a case of acute suppurative otitis media where there was a fistula present. He had seen labyrinth symptoms and vertigo, but had never been able to demonstrate a fistula in an acute case.

Dr. Shambaugh asked himself the question, Shall we operate when there is complete suppression of function, and determined that he would not. Then he referred to the case of the essayist as being a case of suppurative labyrinthitis. He would first speak about the operation.

Abroad, where they are trying to accomplish something, they maintain that we should operate, because it reduces the death rate. They have their statistics of many years' standing in which they did not operate on labyrinth cases, or very rarely. They did not recognize them—they did not know of symptoms that we now speak of as nystagmus and its accompanying symptoms. Dr. Fletcher's opinion is largely their opinion. He is inclined to follow them. What he saw when over there impressed him very much, and what he has seen since returning from there seems to agree with their statistics. The death rate is distinctly lower by operating during the stormy process; in other words, operate on a case that has been diagnosed with certainty as diffuse suppurative labyrinthitis. Bárány and all say, do not operate when it is serous. Sometimes they get brain symptoms. That is, the symptoms lead them to think that there may be a brain abscess. They operate on the labyrinth, and remove it as much as possible, and await results.

With the experience in Europe with a number of cases of

serous labyrinthitis following operation, and with the fact that there is no pus coming from the fistula just now, he is inclined to think that the case is one of serous labyrinthitis rather than suppurative. He has not opened the labyrinth. In other words, while he follows the Vienna school, he also follows his own judgment somewhat. In this case he has seen no indications for an operation. He would not operate on a labyrinth because it was dead. That is a certainty.

In the discussion of what cases we should operate upon, the Vienna men say that we should operate on cases of diffuse suppurative labyrinthitis, because of the great frequency of the occurrence of meningitis and brain abscess in the unoperated cases, and also because they consider the disease more dangerous than the operation. They, on the contrary, consider the operation more dangerous than letting the disease alone, providing you do not do the complete operation. Merely opening into the labyrinth is not the labyrinth operation. They consider, then, the trauma which has been done to the labyrinth infinitely more dangerous than doing nothing, unless you open all three semicircular canals, the vestibule and the cochlea. (The Neumann operation.)

Dr. Beck asked about the other case, the one that he has not operated on, or suggested it. The Vienna school does not approve of that. Bárány especially says to beware of an operation on one ear when the other one is diseased; in other words, he says, you take a chance. This man's hearing is very good. If on either side he should develop very strong symptoms, he certainly would operate—if those symptoms were not serous. If he develops a storm and has any hearing left, or if he responds to turning or to the caloric test in the ear which produces the storm, he would still not operate. But on the first sign of developing intracranial symptoms he would operate.

Dr. Pierce asked if he would operate, in case of intracranial symptoms, whether there was function or not, to which Dr. Fletcher replied: Yes, whether there is function or not.

Replying to Dr. Wilson's remarks on the nomenclature—Bárány objects to that sort of thing. This has been discussed a good deal abroad, and they think they had better retain the present nomenclature, although the speaker quite agreed with Dr. Wilson that it would be very much better to speak of vestibular component.

ST. LOUIS MEDICAL SOCIETY.

OTO-LARYNGOLOGICAL SECTION.

Meeting of March 26, 1913.

Otitic Brain Abscess.

Dr. W. E. Sauer reported a case of an otitic brain abscess in a girl aged nineteen years, with no clinical evidence of mastoid involvement. Otitis media, discharge following paracentesis for one month, ten days after cessation sudden rise of temperature to 101,° pain over left eye, and vomiting, which continued ten days. Examination of ear at that time revealed perforation in the upper posterior quadrant, surrounded by small granulations. No sagging of the upper canal wall, no swelling or pain over mastoid. Kernig's sign present.

Operation.—Mastoid found full of pus; dura over tegmen antri exposed, also lateral sinus; no evidence of intracranial complication found. Improvement for a week. On eighth day following mastoid operation, pulse fell to 60, optic discs slightly congested; ten days after mastoid operation incision of temporal lobe for distance of 2 cm. Abscess cavity found and an ounce of pus evacuated. Patient recovered.

Intraocular Tension in Glaucoma.

Dr. H. Edward Miller reported a case of intraocular tension in glaucoma lowered by injection of Meckel's ganglion. The patient, a woman of fifty-five years, stated that ten years previous she had noticed that her right eye was almost blind and that the left was growing bad. At present time she is totally blind in right eye. She has a severe pansinusitis, accompanied with headaches and increased blurring of vision, in left eye. Following the application of cocain over the sphenopalatine foramina, Schiötz's tonometer demonstrated a drop in tension from 60 mm. to 55 mm. on the right, and from 51 to 37 mm. on the left, within forty-five minutes of its application. Myotics and nasal treatment for a few days brought the tension down to 32 mm. on the right and 26 mm. on the

left. Four months later patient returned with sight much worse. Tension higher than at any time during observation —70 mm. in each eye. Mucous membrane of the nose swollen, but no sinusitis. A course of five injections into the sphenopalatine ganglion of a 5 per cent alcoholic solution of phenol was now instituted, with following results: There was an average immediate rise of 10 mm. and a subsequent average drop of 17½ mm. within twenty-four hours. The minimum drop was 7 mm. The maximum, 28 mm.

DISCUSSION.

DR. W. H. LUEDDE, who observed the case with Dr. Miller, was of the opinion that the case conclusively showed the existence of a relation between nasal disease and intraocular tension. This was noted some twenty years previously by Berger, who showed, among other things, that not only painful attacks of glaucoma could be produced by diseases in the nasal fossæ, but that the application of cocain would give relief, the explanation of which, according to Berger, rests upon the possibility of reflexes being transmitted through the various communicating branches of the fifth nerve. That intraocular tension could be raised by irritation of the superior cervical sympathetic ganglion, the fibers of which accompany the fifth nerve, was demonstrated by Lodato. A commissural connection also exists between the two sides. The immediate rise of intraocular pressure caused by the alcohol injection was probably due to the irritation of the superior cervical sympathetic ganglion. The more permanent and complete nerve blocking, however, should account for the drop in intraocular tension. The commissural connection accounts for the bilateral rise and fall in intraocular tension when only one ganglion was injected. Dr. Luedde reminded of the fact that the short ciliary nerves which are distributed to the choroid and ciliary body, enter the globe posteriorly and come from the ciliary ganglion which receives a root from Meckel's ganglion. Pain impulses passing along these nerves may be blocked at Meckel's ganglion. Pain transmitted over the long ciliary nerves, as from acute iritis, could not be relieved by injection of the ganglion, as they enter directly into the first division of the fifth nerve.

DR. SLUDER reported that the exceedingly great pain that

at times followed injection of alcohol into the ganglion, sometimes lasting as long as two weeks, had led him to use a 5 per cent solution of phenol in alcohol. He also reported the findings of Dr. Otto May, which appeared in the *British Medical Journal* of August 31, 1912, as follows: That alcohol injected into the Gasserian ganglion, as also into the main trunks of the fifth nerve in cats, under direct vision, was followed by almost no destruction of multipolar cells. Alcohol placed in the vicinity of the nerve trunk was destructive for the nerve fiber, but not complete unless the alcohol be placed directly within the nerve proper. Assuming that the sphenopalatine ganglion acts similarly to the Gasserian ganglion under similar circumstances, the experimental evidence, together with the fact that the sphenopalatine ganglion is not easily injected, may explain why the intraocular tension would drop and rise again.

Dr. Bliss stated that there is no direct anatomic way of explaining the pain which frequently is referred to the neck, shoulder, arm, forearm and even down to the hand, in the typical sphenopalatine neuralgia. He thought there was much room for speculation and a great deal more for work, before any definite conclusion on this subject could be arrived at.

New Tonsillectome.

Dr. J. Ellis Jennings presented a new tonsillectome designed to remove the tonsil in its capsule by the Sluder method. It consisted of the French tonsillectome mounted on a pistol-grip, the cutting blade being operated on the "pull" by means of a trigger.

ST. LOUIS MEDICAL SOCIETY.

OTO-LARYNGOLOGICAL SECTION.

Meeting of April 30, 1913.

Nerve Trunk Anesthesia and Carbolization in Nasal Surgery.

DR. GREENFIELD SLUDER presented a paper on nerve trunk anesthesia and carbolization in nasal surgery, in which the writer gave his experiences, extending over a period of three years, with cocain applied over the nerve trunks. His procedure was to place an applicator bearing one drop saturated solution cocain muriate (90 per cent) directly under the posterior tip of the middle turbinate, allowing it to remain fifteen minutes. A second applicator was placed over the internal nasal nerve. For the latter the writer used a smaller applicator which carried only about half a drop of a saturated solution of cocain muriate. An applicator carried blindly into the uppermost anterior aspect of the nose will usually come to rest over the slit through which the internal nasal nerve enters the nose. This nerve, being more superficial, requires less cocain. The anesthesia thus produced, in the writer's experience, gives more than a half hour's time for the performance of the most comprehensive bone surgery of the lateral nasal wall. In addition to the above form of anesthesia the writer resorts to nerve "blocking," using 5 per cent phenol in 95 per cent alcohol, injected by means of a straight needle, for both anterior nasal nerve and sphenopalatine ganglion. For the former the attempt is made to locate the slit from which the nerve emerges, and for the latter it is introduced under the posterior tip of the middle turbinate and carried into the sphenomaxillary fossa, as previously described by the writer. One-half centimeter of the solution is injected. A well-marked analgesia results which prevents to a marked degree the shock of the larger operations at the time of their performance, and the subsequent reaction, especially when the nose is packed.

Plaster Casts of the Sphenoid, Maxillary and Frontal Sinuses.

DR. HANAU LOEB presented some plaster casts of the sphenoid, maxillary and frontal sinuses, prepared to estimate the cubical capacity and superficial area of these sinuses. The work followed a study of the anatomy of the accessory nasal sinuses from the standpoint of their greatest diameters.

The cubical capacity was found to be as follows: Sphenoid, from 0.6 to 11.8 cc., average 4.93; maxillary, from 4.5 to 24.8 cc., average 12.94; frontal, from 0.9 to 8.2 cc., average 3.58.

Superficial Area.—Sphenoid, from 2.4 to 28.2 sq. cm., average 15.92; maxillary, from 12.1 to 52.3 sq. cm., average 31.68; frontal, from 5.5 to 32.3 sq. cm., average 15.34.

ST. LOUIS MEDICAL SOCIETY,
OTO-LARYNGOLOGICAL SECTION.

Meeting of May 28, 1913.

The Endonasal Route of Attack in Hypophyseal Tumor Cases.

Dr. W. E. Sauer presented a paper under the above title, in which he reviewed the literature to date, and more especially that of Hirsch of Vienna, according to whose method he operated an acromegalic aged thirty-two years. The patient had been under observation for a period of one year, during which time very little differenec in any condition, mental or physical, was noted, with the exception of a marked decrease in the vision of the left eye. Vision right eye was 15/12; left eye, 15/200. Several months after the sella turcica decompression operation, which was done under local anesthesia, the patient's vision improved to the normal in right, and in the left to 17/40. The discussion brought out that the danger of subsequent infection and meningitis was more apparent than real, and that tearing of the mucous membrane need not be considered so seriously, especially in view of the fact that there are two permanent sphenoidal ostia through which infection could take place.

The program further consisted of a general discussion of:

(a) Form of initial incision and mode of anesthesia in submucous resection of the nasal septum.

(b) "Cut or cauterize" the inferior turbinate.

V-Shaped Cautery Tip.

Dr. Sluder showed a V-shaped cautery tip, with which, besides cauterizing, one could remove a wedge-shaped strip of mucous membrane from the entire length of the inferior turbinate, much as a small spoke-shave would.

ABSTRACTS FROM CURRENT LITERATURE.

I.—EAR.

The Radical Mastoid Operation Under Local Anesthesia.

KULENKAMPFF (*Beitrage zur klinischen Chirurgie*, Vol. 83, p. 546), in a very lengthy article, gives a detailed account of the method by which he has carried out thirty-two radical mastoid operations. All of these cases were completely successful from the standpoint that the patient did not suffer the slightest pain or inconvenience.

The technic is as follows: The patient is first given two preliminary injections of scopolomin morphin, in the usual doses. The skin behind and in front of the ear is wiped off with benzine, but not otherwise sterilized. The first point of injection is just above the concha; a 1 per cent solution of novocain, to which has been added a few drops of adrenalin, is freely injected in the loose tissue just above the periosteum, but not under it, in a backward direction, following the insertion of the concha. Without withdrawing the needle the same solution is injected in front of the ear. The second point is behind the ear, opposite the external canal. The injection is first made upward to meet the first injection and then downward toward the insertion of the s. c. m. muscle. If there is reason to believe that the process is extensive or that the mastoid tip is large, a second point is selected one inch behind the last named point and the injection made up and down, following along the hair line. The third principal injection point is at the posterior border of the external bony canal, the needle being inserted in the fold behind the ear and shoved in perpendicularly until it is under the skin of the external canal. For this purpose a 2 per cent solution is employed. When the skin of the canal has been thoroughly infiltrated, the Neumann injection into the junction between the cartilaginous and bony canal is made and the fluid slowly forced into the middle ear between the skin and the bony surface of the canal. If a perforation is present, a pledget of cotton soaked with 20 per cent alypin solution is placed in the mid-

dle ear. The field is now prepared for operation, and within ten minutes from the start the patient is ready. The author lays great stress on the fact that the patient should be made thoroughly comfortable and that the position of the head should be so arranged that the subsequent manipulation will not tire the patient.

The subsequent steps of the operation are carried out in the usual way, the author prefers to make his plastic before breaking down the bridge. The choice of chisel or forceps depends on the condition of the bone and is largely influenced by what the patient seems to stand the best. Acute mastoids, fresh attacks of old processes and intracranial complications are contraindications for this method. *Horn.*

The Bier Passive Hyperemia in Nonpurulent Affections of the Middle Ear.

STEIN (*Archiv. für Ohrenheilkunde,* Vol. 86, p. 92), in two articles, has made an exhaustive study of the physiologic and clinical features of the Bier passive hyperemia in nonsuppurative affections of the middle ear. He draws the following conclusions:

First, the employment of Bier's method possesses in nonpurulent affections of the middle ear scarcely the meaning of a well established therapeutic procedure. In special cases, however, it can perhaps be employed to assist the other therapeutic measures.

Second, subjective sensations proceeding from the ear, as regards their genesis, can be distinguished by the aid of the passive hyperemia to a certain extent. In cases in which the ear noises are diminished by the application of the band, the cause of the noises should be found in the effect of the pulsatory movements of the liquor cerebrospinalis. Such sounds arise especially in an increased blood pressure, whether caused by physiologic or pathologic processes, since it calls forth an increase of the pulsatory movements of the cerebrospinal fluid. Under all circumstances it is demanded, in cases in which the rubber band produces a lessening of the ear sounds, that an accurate internal examination should be made. Increased sensitiveness of the nervous system and pathologic changes in the sound conducting apparatus lead to an increase in the per-

ception of the impulse proceeding from the liquor cerebrospinalis, and in this way to an origin of the ear noises.

These features can also in slightly elevated or even in normal blood pressure give rise to subjective sensations in the ear. *Harris.*

Otitis Media Acuta Due to the Bacterium Pneumoniæ Friedländer, Its Prognosis and Treatment.

ZANGE (*Archiv. für Ohrenheilkunde,* Vol. 89, p. 1) has had two fatal cases of acute otitis media due to the Friedländer bacillus. He describes these at length and makes the following summary:

From the cases occurring in the literature and our own observations, otitis media acuta due to the Friedländer bacillus, like the Friedländer pneumonia, presents a characteristic clinical picture, the peculiarities of which are based on the presence of the Friedländer bacillus, towit, in the enormous reproductive capacity and marked mucus production of the bacillus in the organism, associated with slight local reaction of the tissues. The middle ear secretion is from the beginning mucopurulent, microscopically very rich in mucus and bacillus, poor on the other hand in pus and fibrin. The Friedländer otitis shows often from the beginning an insidious character without markedly pronounced local symptoms, sometimes with and sometimes without fever. It is wont to run a protracted course. It leads further to severe complications of the mastoid, the adjoining parts and the intracranium, with often fatal outcome.

Complications in the same way can develop insidiously and without clearly marked symptoms. The Friedländer otitis demands, on this account, particular care in observation, and the prognosis must under the circumstances be regarded in no case as favorable. On this account it is also important as early as possible to make the diagnosis of Friedländer otitis, and thus by an early operation to be able to check its advance or the fatal results of severe complications.

To this end it is recommended that in every bacteriologic examination of the secretion from the ear, the staining of the capsule should be the principal thing. For this thionin is admirably adapted, which, as is already known, shows very well the capsule of the streptococcus mucosus. *Harris.*

Sudden Deafness From Scarlet Fever Otitis, and the Recognition of Otitis Interna Serosa.

MANASSE (*Archiv. für Ohrenheilkunde,* Vol. 89, p. 146) gives a valuable contribution to this important subject. Up to the present any accurate description of the microscopic changes occurring in the middle and inner ears as a result of scarlet fever otitis is lacking. Politzer in the last edition of his book reports no case microscopically examined directly after the onset of the deafness. Manasse describes two cases occurring in his practice, of scarlet fever deafness, which recovered as far as the general condition was concerned, although the hearing was not improved, and a third case which went to autopsy. Of this latter case he has made a careful microscopic study and gives the findings, together with illustrations.

The alterations which took place in the middle and inner ear he summarizes as follows:

On both sides there was a typical acute necrosing otitis media scarlatinosa. On the left side there was a total destruction of the ligamentum annulari and of the stapes; purulent inflammation of the membrana tympana secundaria with a small fistulous perforation; otitis interna purulenta diffusa with ostitis acuta of the labyrinth capsule. On the right side there was total destruction of the membrana tympana secundaria; purulent inflammation of the ligamentum annulari without perforation; otitis interna purulenta circumscripta in both windows; otitis interna serosa diffusa of the membranous labyrinth; beginning acute ostitis of the bony semicircular canal; degenerative atrophy of the cochlea.

There are a few cases in the literature of microscopic examinations of the inner ear in scarlet fever otitis. None of these, however, were made immediately after the onset of the attack. Manasse is of the opinion that the perforation through the left round window was the cause of the sudden deafness in his case. He raises the interesting question whether in the first two cases, clinically but not anatomically examined, the pathologic condition was not the same. In all three cases we have to do with grave scarlet fever diphtheria, double diphtheria, scarlet fever otitis, characterized by a necrosis of the soft parts and of the bone. All three cases show the same degree of sudden complete deafness following hearing rela-

tively well preserved. Each of the two cases which are alive has passed over into deafmutism.

In view of these facts, he is of the opinion that the anatomic changes were similar in all three cases, although he would not claim that in all cases of scarlet fever deafness the process is the same and that there cannot be an involvement of the auditory nerve. All cases of complete deafness following scarlet fever otitis are not necessarily permanent, as is shown by a case which he reports, where the hearing following a radical mastoid operation returned, and the patient is now able to hear the conversational voice at a distance of from five to six meters.

The article concludes with the anatomic findings in the case of a patient seventeen years old, who had suffered for fifteen years from a purulent affection of the middle ear, the result of an attack of scarlet fever. The case was operated upon for mastoiditis and brain abscess, and the findings in the middle and inner ear are accurately described. Those of the inner ear are especially interesting. They include an extensive atrophic degeneration of the nerve elements, especially of the organ of Corti, the ganglia spirale, and of the fine terminal branches in the cochlea. He believes that all the changes in the middle ear and labyrinth must be ascribed to a chronic inflammation of the soft parts and of the bone, which has run its course. He admits, however, that the interval of time elapsing does not allow a positive opinion, but he is inclined to believe that in this case, and in any other similar case, we are warranted in speaking of a healed otitis internus serosa.

Harris.

The Relation of the Ostium Tympanum of the Tubæ Eustachii to the Fundus of the Cavum Tympani, as Well as to the Foramen Rotundum and the Foramen Ovali.

ZYTOWITSCH (*Archiv für Ohrenheilkunde,* Vol. 89, p. 280) has by means of a specially constructed apparatus been able to make accurate measurements on the dry skull of the landmarks in question. Among the interesting deductions from his measurements which he makes are the following:

That the ostium tubæ eustachii in a horizontal position of the skull is only 3.7 mm. higher than the floor of the cavum tympani and almost on a level with the middle of the niche of the foramen rotundum; the foramen ovali is 3.7 mm. higher

than the ostium tubæ eustachii. As compared with the antrum mastoidæ, the tubæ eustachii lies considerably deeper. By inclination of the skull fifteen degrees forward, these relations change and the tubæ eustachii becomes lower. In general, the ostium tubæ eustachii has a position extremely favorable for the drainage of fluid from the middle ear toward the nasopharynx. As a result of these measurements he draws the following conclusions:

First, (a) the tubæ eustachii functionates as a ventilation canal for the middle ear; (b) if there is secretion present in the tympanic cavity, the eustachian tube offers a means of escape for it. Second, in the presence of symptoms of acute otitis the patient must be instructed to bend the head as far as possible downward and to the shoulder on the healthy side. Forcible respiration through the nose has a distinctly beneficial effect in restoring the patentcy of the eustachian tube, thereby assisting in the removal of the secretion from the middle ear. *Harris.*

A Case of Erosion of the Sinus With Embolus and Pyemia, With a Fatal Result.

SCHLAGEL (*Archiv. für Ohrenheilkunde,* Vol. 90, p. 45). The patient was a man of 29 years, operated upon for chronic middle ear suppuration. The operation revealed the sinus lying far forward. As the result, at the outset a slight wounding of the wall took place. Two weeks after the operation a severe hemorrhage took place, and the next day, when the dressing was removed, an embolus through the vein took place. In spite of a ligation of the jugular, it was impossible to control the development of the pyemic process, and the patient died a month after the operation. The author considers some of the questions raised by his case. Erosion of the sinus is not an exceedingly rare occurrence. Twenty cases up to 1905 were reported in the literature, of which only six came to operation. An embolus was the most interesting phenomenon. He dwells on the fact that it could have taken place and not been followed by a fatal result.

He considers especially the three questions as bearing upon the fatal result: first, of the exposure of the sinus; second, the ligation of the jugular; third, the failure to clean out the bulb. He admits the unfortunate result of having bared the

sinus, but insists that under the circumstances it was impossible to avoid it. There was, in his opinion, no occasion for a ligation of the jugular at an earlier time. As regards the bulbous operation, there appears to be a divided sentiment. Personally he doubts if the operation would have had any effect, however. *Harris.*

The Whispered Voice and Conversational Speech in Their Relation to One Another.

JULIUS VEIS (*Archiv. für Ohrenheilkunde,* Vol. 90, p. 200) has made an extensive comparison of the whispered voice and the conversational speech in various forms of ear trouble, and as a result of these investigations he is of the opinion, first, that the testing of the spoken voice as well as the whispered voice in every case of marked lowering of the hearing (whispered voice under one meter) is to be made from a diagnostic, therapeutic and prognostic standpoint. The testing with the whispered voice alone does not give a satisfactory picture of the acuteness of hearing. Secondly, in otosclerosis and in many cases of pure middle ear suppuration, the conversational voice (deep tones) is not heard much further than the whispered voice (high tones), while, on the other hand, nervous hard hearing and in the exudative middle ear processes, the conversational voice will be heard much better, comparatively, than the whispered voice. Thirdly, when the conversational voice is markedly better than the whispered voice, there is a decidedly better outlook as regards success in treatment. Fourth, the improvement of the hearing by air inflation many times affects only the conversational voice, while the whispered voice is only slightly or not at all.

Harris.

Plastic Operation for Congenital Malformation of the Hearing Organ.

KOSOKABE (*Archiv. für Ohrenheilkunde,* Vol. 90, p. 214) reports two cases operated upon for deformity of the auricle and external auditory canal. Kaufmann's observations are referred to in such cases: First, that the tuba eustachii is for the most part irregular. Second, the bony wall of the auditory canal is lacking. Third, the temporal bone is often deformed. Fourth, the drum membrane and the tympanum

are not present. Fifth, the labyrinth windows and the stapes are for the most part normal. Sixth, men and women were equally affected.

The indications for operating in such cases he gives as follows: First, when it is possible to show the existence of any hearing; second, when stimulation of the vestibular apparatus is positive; third, when a cosmetic operation is urgently desired.

As a result of his study of the two cases, he gives the following results: First, by operation the lower tone limit is extended; second, the upper tone limit is not influenced; third, there was no sound hiatus, as in Bezold's case; fourth, bone conduction is no longer than normal, and Rinné was negative, both before and after the operation; fifth, the disturbance of the hearing was similar to that produced by obstructions to the sound conducting apparatus; sixth, in both cases there was an improvement in the hearing after the operation; seventh, the middle ear and the ossicles were lacking, but the mastoid cells were well developed; eighth, the parotid gland was found lying in the space of the auditory canal; ninth, the direction of the sound was correctly given in Case 1, but not in Case 2. *Harris.*

The Acoustic Nerve and Salvarsan.

GELLE (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* October 19, 1912). After referring to the statistics of Benario, which show that in 14,000 cases treated by salvarsan there were 126 cases of neurorecidive, and of these 126 cases, 62 had involvement of the acoustic nerve; and of Valentin, who reports 45 cases of lesions of the auditory nerve after the injection of salvarsan, he reports three cases occurring in his own practice.

The first was a case of recent syphilis which had been treated by salvarsan with satisfactory results. Three injections were given at intervals of a week. Fifty days after the first injection deafness appeared in the left ear. Another injection of salvarsan was given. Three days later deafness in the right ear resulted. The deafness in the right ear entirely disappeared after seven days, but persisted in the left ear. The Wassermann in this case remained positive.

The second case was a young man of twenty-four years of age, where the initial lesion had been contracted two months

before. Sixty-three days after the first injection and forty-six days after the last injection (three injections were given in all), a right facial paralysis of peripheral origin resulted, followed by paralysis in the right cochlea and the vestibular nerve. Two additional intravenous injections of salvarsan were given after the development of the paralysis, but without effect. Here also the Wassermann reaction remained positive.

The third case was a man of twenty-seven years of age with a history of syphilis of one year's duration. Salvarsan was given for the secondary skin lesions. Forty-two days after the last injection (four injections were given), there was a bilateral deafness (cochlear and vestibular nerves). On the right side the deafness disappeared in about fifteen days. It persisted on the left side.

The author feels that in these three cases there is a distinct causative relation between the salvarsan and the deafness. The toxic action of the salvarsan would not be affected by the further use of the drug. On the other hand, a mechanism due to the toxic action of insufficient trepomenes would probably be relieved by the further use of salvarsan. As will be noted in these three cases, the use of salvarsan was continued without avail. He calls attention to the curious course of the deafness. The ears were affected after an interval of some days, then at the end of eight days hearing reappeared in the ear last affected. The size of the dose seemed to have no effect, as in one case small doses were used and in another large doses. In all the cases the Wassermann reaction remained positive, and lumbar puncture always showed an albuminous fluid with pronounced leucocytosis. Two of the cases had shown a great intolerance to mercury. As a result of his experience with these three cases, the author favors the employment of mercury and the iodids in any syphilitic involvement of the ear. For nasal and laryngeal lesions he advises 606. *Harris.*

Dilatation of the Jugular Bulb, Filling the Middle Ear and Part of the External Auditory Canal.

LAMMOIS (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* October 12, 1912) refers briefly to the anomalies of the jugular bulb described by Zuckerkandl. At

times the bony wall is reduced to paper thinness. In a case of Odenius it formed a sufficient prominence to hide the round window and the promontory. The bone can even be so thin as to give rise to true dehiscences.

In a number of cases the jugular bulb has been wounded, in the auditory canal, usually in the course of the paracentesis of the drum membrane, sometimes in the extraction of foreign bodies, and again in the removal of the ossicles.

Lammois has recently seen a case of marked dilatation of the bulb. The patient was a woman 50 years old, suffering from suppuration of the ear, which had existed for seven years. There was an offensive odor and polyp-like granulations which suggested the possibility of cancer. Under general anesthesia the auricle was thrown forward. Inspection then showed that the membranous auditory canal was lacking, and that in the canal there was a small violet-like growth the size of a small walnut. The bony canal was found to be enlarged, with irregular borders, giving the impression of a condensing ostitis. The mastoid cells were healthy, and neither pus nor granulation were found in the antrum. Further examination of the mass showed it to be a bluish color, the appearance of a large vein, and it was easily reducible in the middle ear. There was no pulsation under the finger. A Pravaz gave black blood. The diagnosis was made of dilatation of the jugular bulb, which filled the middle ear and formed a prominent projection in the external auditory canal.

The author believes that the venous hernia was not due here to a spontaneous dehiscence or to an erosion of the blood current, but rather to a progressive caries of the hypotympanic recess. *Harris.*

A Contribution to the Study of the Reeducation of the Hearing.

RICHEZ (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie*, November 9, 1912). The failure of all forms of treatment for otosclerosis is generally recognized. One cannot prevent the progress of the disease, but he can strive to awaken a dormant organ and reeducate it by appropriate exercises, just as one restores suppleness by means of massage and anchylosed articulation. The idea of reeducation is not new. Urbantschitsch was the first to demonstrate this by employing an accordion, but this method was not

satisfactory. Zund-Burguet was the first to see the desirability of employing for this purpose the human voice, because of its richness in the low harmonics. He showed, however, that for manifest reasons it cannot be employed in the education of the deaf, and accordingly has devised an apparatus to take its place.

The apparatus is called the electro-vociphone, and is capable of reproducing the sounds which the human larynx can give forth. The agent is a number of layers of cotton, making a true artificial larynx corresponding to the three registers, the low, medium and high, and put in vibration by a current of from four to six volts. The intensity of the sound can be instantly varied according to need, and if one of the two ears is more sensitive than the other, it can be treated apart from the other.

The author has employed the apparatus with surprising results, and is thoroughly convinced of its value. He gives a report of six cases in his practice where it has been used with success. In conclusion he raises certain important questions, first, as to the permanency of the cure. His first case contracted sore throat with a temporary reduction of the hearing. With the disappearance, however, of the angina, the hearing returned and became as good as formerly. In the experience of all who have used it, the results are maintained at the end of three or four years.

In answer to the question, "What is the variety of deafness suitable for the treatment?" he replies, "Especially cases of sclerosis where the deafness has come on insidiously or as the result of confinement, or grippe, or of an infectious disease." It is indicated also in cases of adhesive otitis and in chronic middle ear catarrh the result of nasopharyngitis, although in these cases the results will be much better if treatment of the nose and throat is undertaken at the same time.

Harris.

Personal Results Obtained in Otology by the Aspiration of Bier.

ERNEST BOTELLA (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie*, November and December, 1912). The use of the Bier band in otology has been attended with serious consequences. On this account, Botella has substituted aspiration for it. For this purpose a Politizer bag or

syringe can be employed. He makes use of curved glass tubes having an olive-shaped head at the end. These are of different sizes and are introduced deeply into the canal. A rubber tube connects the canula with a strong rubber bag. By these means he has obtained most satisfactory results. In a series of ten cases of ear suppuration which he reports, four were caries of the attic, in which although a cure was not obtained there was considerable improvement; two were cases of caries of the attic which were cured; two were cases of acute otitis media with mastoiditis, which were cured; one was a case of chronic otitis media, cured; and one was a case of large polypus of the tympanum, where all treatment was discontinued at the second visit.

Aspiration has the double advantage of removing the secretion from the tympanum and the adjacent cavities, and secondly, of producing an active hyperemia in the parts affected. Its curative action depends upon a phagocytosis and a reconstitution by an increase of the vitality, rather than by accumulation of antitoxin as produced in the ordinary method of Bier.

He reaches the following conclusions: First, that the direct aspiration of the auditory canal is the best means of evacuating the secretions from the middle ear; second, in order to avoid the inconvenience which can be produced by pressure on the walls of the canal, canulæ are to be recommended, carried well into the canal; third, the perforation of the drum is to be carefully watched in order that it may be enlarged if it has a tendency to close before the diseased process is cured; fourth, although aspiration can be employed without danger in all cases, the results are most favorable in simple acute otitis media with mastoiditis, and simple chronic otitis media; fifth, aspiration for the removal of granulations and small polypi is superior to the curette, because its action reaches regions where the curette cannot touch; sixth, it can be tried in cases of adhesive otitis and generally in all cases where it is thought necessary to counterbalance the effects of the external pressure upon the tympanum and the ossicular chain; seventh, the treatment should last from ten to twelve minutes at the maximum, it should be stopped if blood follows, and should be carried out gently in order to prevent nausea or pronounced vertigo. *Harris.*

Acute Otogenous Meningoencephalitis.

G. ALEXANDER (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* November and December, 1912) is of the opinion that meningitis is not a distinct entity, but that there exist different forms of meningitis; as, for instance, traumatic, ophthalmic, rhinologic., etc., each different distinctly from the otitic.

He divides meningitis of the ear into five groups: First, meningitis in acute suppurative otitis media without other complications; second, meningitis in chronic suppurative otitis media without complication; third, meningitis in cerebral abscess of otogenous origin; fourth, meningitis in thrombophlebitis of otogenous origin, and extradural abscess; fifth, meningitis in suppuration of the labyrinth. He further divides meningitis into early and late meningitis. He then gives the report in detail of seven cases of meningitis, all cured by operation. The first case was a child of four and one-half years of age, suffering from serous meningitis following acute suppuration of the middle ear; the second case was also a child of two years, where the meningitis followed a chronic suppuration of the middle ear. Following the operation the patient was unconscious for two weeks. The third case was a man of forty-two years of age who had suffered from acute suppuration of the left ear and osteoperiostitis of the left mastoid and serous meningitis. An anthrotomy was done and the dura exposed and incised in the middle cerebral fossa. The fourth case was a man of forty-four years of age with chronic suppuration of the middle ear, cholesteatoma, purulent (?) circumscribed meningitis. Case five was a child of nine years of age suffering from bilateral suppuration of the middle ear with cholesteatoma and acute meningitis, a cloudy but sterile spinal fluid. Case six was a chronic suppuration of the middle ear with cholesteatoma, serous meningitis, circumscribed purulent labyrinthitis; radical operation, and exposure of the dura. Case seven was a man of twenty-four years of age with chronic suppurative otitis media, cholesteatoma, suppurative labyrinthitis, acute purulent meningitis, paralysis of the external ocular motor, paralabyrinthitis, osteomyelitis of the pyramid and the base of the skull in this region; radical labyrinthine operation, a traumatic fracture at the base of the skull.

Harris.

The Residual Ear.

DAVID G. YATES (*New York Medical Journal,* December 7, 1912) defines as residual a middle ear which has been the seat of a suppurative process which has ceased. There may be either a perforation of the drum in these cases or an intact membrane. He is of the opinion that a considerable number of cases of diving or surf-bathing, where an acute inflammation of the middle ear has been set up, belong to this class, and advances the interesting theory that many unexplained drowning accidents, in his opinion, are due to vertigo, the result of the entrance of cold water into an unprotected middle ear.

He regards as an important part of treatment preventive measures, and advises opening the mastoid when in acute cases, after free incision of the drum, postauricular tenderness persists for two or three days and there is a rise in temperature. For recent perforation, the application of a small disc of gutta percha tissue over the opening is recommended.

There may exist a necrosis in the middle ear without discharge. Once a year perhaps a few drops of pus will be evacuated. Epidermization of the edges is the chief reason for nonclosure of the apertures in the membrane. The prospect of healing of the drum is in inverse proportion to the size of the perforation. Tinnitus or marked deafness are sometimes relieved by division of bands or deposits of fibrous tissue. The writer very properly states, however, that the tendency of this adhesion is to reform. He urges the danger of the patients passing from observation before the drums are entirely healed. *Harris.*

The Radical Mastoid Operation—Some Essentials for the Proper Healing of the Wound Cavity.

ROBERT L. LOUGHRAN (*New York Medical Journal,* December 21, 1912). The complete removal of all areas actually diseased, or that might retard the development of healthy granulations, the establishing of satisfactory drainage, and the producing of the means of easy observation and local treatment, give the best opportunities for the rapid and permanent healing of the wound.

The author regards thorough curetting of the mouth of the eustachian tube as essential to the success of the operation.

(This is the procedure of most otologists in this country, but the Vienna school has in recent years abandoned it, leaving the tube entirely undisturbed.)

The effect of any constitutional disease upon rapid healing is dwelt upon. The author explains at length the physiology of healing of the operative cavity. Due to the greater blood supply, granulations form more rapidly in the antral wound than they do in the tympanic cavity. The development of the epidermal layer is of especial importance and depends to a large extent upon the way that the original flaps are placed. Deafness in chronic middle ear suppuration is usually due to involvement of the sound conducting apparatus. Great care is to be exercised in the preservation of the stapes on that account.

Posthealing contraction of the scar tissue over the stapes cannot be prevented, and it is this which often accounts for the frequent flagging of hearing some time after operation. Another influence as regards the preservation of the hearing is the auditory canal. While the opening needs to be enlarged, it should be done in accordance with its requirements as the conductor of sound waves. The writer properly states that the radical operation does not restore hearing to the normal, nor yet does it destroy it.

Finally, attention is called to the cosmetic results, which, in his opinion, depend upon three things: the appearance of the posterior incision and that of the concha, and the position of the auricle. The first is secured by care in making the incision and by avoiding traumatism to the edges during operation. Depression can be avoided by care in placing the incision in a location where it may have a foundation for healing; that is, on the skull surface, posterior to the margin of the wound cavity. *Harris.*

Progressive Deafness—Systemic Factors in the Etiology.

J. A. Stucky (*New York Medical Journal,* December 28, 1912) considers progressive deafness under the two divisions of nonexudative otitis media and otosclerosis. He believes that these diseases are, more than any other ear trouble, a local manifestation of the systemic condition. The subjective symptoms of otosclerosis differ very little from those due to nonexudative otitis media, except in intensity. Giddiness or vertigo

occur less frequently, and seldom in aggravated form. He has collected a series of cases typical of the two diseases, which he has been able to keep under observation for from five to fifteen years, and to work out a line of treatment and method of living which has given results that justify him in the conclusion that in order to bring about favorable results it is the systemic rather than the local factor in the causative relation of these conditions with which we must deal.

The majority of cases of these diseases, in his opinion, give evidence of disturbances and faulty metabolism, indicated by the presence of endican or phosphates in the urine, or there are present indications of rheumatism. The next systemic condition most frequently observed was increased blood pressure or arterial sclerosis.

He believes that when the cure for rheumatism and arterial sclerosis is found, we shall also have solved the cure of non-exudative otitis media and otosclerosis. *Harris.*

Vertigo—A Clinical and Therapeutic Study.

RICHARD LAKE, London (*New York Medical Journal,* January 4, 1913), divides the causes of vertigo as follows:

1. Peripheral causes. (a) Chronic progressive middle ear deafness. (b) Hemorrhage into the labyrinth, and embolism. (c) Traumatism.
2. Aural vertigo due to altered state of the blood pressure. (a) Increased blood pressure. (b) Decreased blood pressure.
3. Aural vertigo due to systemic causes. (a) Leuchemia. (b) Occasional. (c) Specific. (d) Anemic (cerebral anemia).

One of the most common causes is chronic progressive middle ear deafness. This form is of purely peripheral origin. Operation should be tried, if local and systemic measures fail. The results are immediate and certain relief. In a series of some seventy cases presenting this condition for treatment, it has been found necessary to operate on only fourteen. In each case the operation was successful as regards relief of the vertigo.

Emphasis is to be laid upon antiseptic rather than aseptic surgical measures.

In traumatic cases the vertigo rarely lasts more than six months. General measures of treatment are usually fairly

successful. Two of the common but little recognized causes of this form are plugs of cerumen and the effects of violent nose-blowing. A characteristic feature of these cases is the persistence, after the disappearance of the vertigo, of an inability to walk in the dark.

The second great group of causative factors is altered blood pressure. Here also the lesion is peripheral. A peculiar feature in these cases of arterial sclerosis is the tendency of the attacks to come on in the early morning. Cases in which the blood pressure is below normal show a greatly increased vertigo on rotation. Cases due to leuchemia or pernicious anemia are not amenable to treatment. Among the most frequent of occasional causes is gouty dyspepsia. The author is of the opinion that operation is seldom needed, but when required is practically free from any danger to the life of the patient. *Harris.*

Otology From a Medical Point of View.

H. N. SPENCER (*New York Medical Journal,* March 1, 1913), in a short but philosophic paper, emphasizes the importance of the otologist establishing a close connection with the laboratory. His view that wholly inadequate consideration is given to the efficiency of internal medicine in the treatment of diseases of the ear and of the nose will be generally accepted.

He urges more frequent examination of the urine and of the blood, as well as inquiries into the abnormalities of digestion, etc. *Harris.*

II.—NOSE.

Connection of the Sexual Apparatus With the Ear, Nose and Throat.

VIRGINIUS DABNEY (*New York Medical Journal,* March 15, 1913) gives an excellent review of the literature on this subject and the history of eight cases occurring in his own practice illustrating the relation in hand.

His conclusion is that "many cases escape diagnosis in whom the sexual element is the contributing if not the exciting cause of many of the common nasal and aural abnormalities of function, partly for the reasons stated in his paper, partly from the lack of time needed to study intimately these cases, and partly from failure of the patients to give their full confidence." *Harris.*

Disease of the Nasal Accessory Cavities.

SEYMOUR OPPENHEIMER (*New York Medical Journal*, March 15, 1913) agrees in the main with the conclusions of most rhinologists of today. His view, however, that "combined frontal and ethmoidal suppuration, with more or less polypoid degeneration of the middle turbinal, require an external radical operation for a permanent cure," would be held by most men to be extreme. This statement he qualifies, however, by adding that where there is free drainage and no signs of ill effect in the general health, the risks of the operation more than counterbalance the advantages.

He rightly states that the aim of any radical procedure is the obliteration of the affected cavity, and that if this is not accomplished, reinfection is always a possibility. *Harris.*

Treatment of Nasal Necrosis by Means of Radium.

BROECKAERT (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie*, July 27, 1912) gives a report of four cases treated by radium with uniformly good results. He employs one cm. of the bromid of radium with a radioactivity of 500,000. It can be applied either according to the socalled destructive method which produces a necrosis of the diseased portion and subsequent cicatrix, or by the modified method which produces a slow and gradual transformation of the affected tissues.

He favors the latter method in most cases, especially in the ulcerated form of lesion, the time of exposure varying according to the extent of the lesion and the rapidity with which one wishes to produce results. The method is characterized by the remarkable uniformiiy of the results and the complete freedom from reaction.

In his hands it has given uniform success, and he regards it as the ideal method of treatment for necrosis of the nose.

Harris.

A New Contribution to the Treatment of Ozena by Functional Nasal Reeducation.

ROBERT FOY (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie*, August 10, 1912) divides the treatment into two stages. The first is a preparatory stage, lasting from eight to fifteen days and consisting, first, of the injection into each nostril night and morning of hydrogen

peroxid; second, waiting three minutes before blowing the nose; third, the introduction of boric acid powder in the nostrils by means of an autoinsufflator; fourth, waiting five minutes before blowing the nose; fifth, lavage of the nose with a liter of hot water in which is dissolved a mixture of salt, glycerin and tincture of iodin; sixth, five to ten minutes breathing in the open air; seventh, internal treatment with iodid of potash.

At the end of this time the special treatment is begun, which consists, first, of pulverization with dry air under a pressure increasing from zero up to five kilos; second, massage of the nasal mucosa, especially the head of the middle turbinate and the upper region of the septum with cotton rolled upon an applicator; third, breathing exercises, first with one and after with two nasal olives introduced into the nostrils of the nose, moist air being introduced at intervals. The treatment lasts a half an hour.

During the second stage of the course of treatment, irrigations of iodin are discontinued. Twenty to forty seances are necessary, at first daily, and later every other day. In addition to the treatment with the dry air, three to four times during the day for ten minutes simple respiratory exercises are practiced. These have the object of establishing the nasal respiration and increasing the vitality of the mucous membrane of the upper air passages. Three exercises are given, the first is purely nasal, the second, costodiaphragmatic. At the end of the treatment the patient is usually cured. There can be relapses of a minor degree, which are relieved by reeducation and more careful respiratory and hygienic regime. If the patient is not cured but only benefited, a second course of treatment, lasting three or four months, is in order. Only very rarely is a third course necessary in order to obtain a cure.

At times, after the patient is well, the use of paraffin is desirable but not necessary. The author insists that ozena is not a specific affection, but a trophic disease affecting the passages of the upper air tract and causing a suppression of the functions of this mucosa. Air is the physiologic stimulant of the mucous membrane exposed to the air, just as food is the stimulant of the mucous membrane of the digestive tract. The treatment outlined restores to the nasal mucous membrane its normal physiologic stimulant, air.

The author concludes by giving the report of a number of cases of ozena which have been cured by the method.

Harris.

Fractures of the Nose.

Garel (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* August 31, 1912) dwells upon the importance of the immediate correction of all fractures of the nose, as a means of avoiding extensive operative measures later on.

He reports a case recently under his care where he employed sheets of gutta percha for an external retaining splint, with excellent results.

(Abstractor: As is well known, gutta percha has been used for this purpose in America for a number of years.)

Harris.

The Treatment of Nasal Synechia by Means of Mica Splints.

Moure (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* October 26, 1912). In Moure's hands the thin lamellæ of mica have proved more satisfactory than any other method employed for this condition. He has used it in many severe cases, with excellent results.

It is so thin that it does not cause any irritation and does not interfere with the normal secretions. It is easily sterilized, standing a high degree of temperature. It also permits of being bent. All that is necessary is to give the mica the form that it should have inside of the nose. *Harris.*

The Sphenochoanal Polyps.

Kubo (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* March and April, 1913). The result of Kubo's investigations are summed up in the following conclusions:

First, that solitary choanal polyps can proceed in the same way as antrochoanal polyps or maxillary choanal polyps, and have their origin in the sphenoidal sinus, especially in the vicinity of the lower part of the ostium. To these polyps he gives the name of sphenoidal or sphenochoanal polyps.

Second, in the early stages of their formation these polyps are found in the sphenoidal sinus and are discovered accidentally when the sinus is opened, so they are characterized as a polyp-like thickening on the upper choanæ border just

below the ostium. This latter phenomenon is an important objective symptom of chronic sphenoidal sinusitis.

Third, in the later stage of their formation, sphenochoanal polyps can become so large that they totally obstruct the choanæ. This is why the differential diagnosis from antrochoanal polyps is very important from a therapeutic standpoint. It is necessary before operation to make a careful examination, because the sphenochoanal polyps can resemble closely the nasopharyngeal fibroma, as the result of their short pedicle and the situation of their base.

Fifth, in Kubo's opinion the majority of polyps spoken of as choanal are in effect sphenochoanal.

Sixth, in sphenochoanal polyps the sphenoidal ostium is generally very large.

Seventh, simple removal with the snare is not sufficient as a radical operation for sphenochoanal polyps. It is necessary at the same time to treat adequately the source of their origin, that is to say, the sphenoidal sinus itself. *Harris.*

III.—PHARYNX.

The Factor Intoxication in the Genesis of Adenoid Vegetation.

FISTRE (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* July 6, 1912). An unusually thoughtful and suggestive contribution to the etiology of adenoids. The article should be read in full, but his conclusions are as follows:

Hyperplasia of the lymphatic tissue of the pharynx constitutes a visible evidence of a defensive action of the infantile organism against a chronic intoxication, whether inherited or acquired.

It forms an integral portion of a complex syndrome, often discrete, i. e., the rachitis, affecting at first the bony marrow, then almost immediately afterwards the lymph organs, and later only the kidneys, spleen and the other internal organs.

In a case where the organism of the infant is profoundly affected, adenoidectomy alone gives indifferent results as regards the later somatic development. To accomplish a complete cure, not merely are appropriate respiratory exercises necessary and a general hygienic and dietetic treatment, but also an actual cure by means of polyglandular opotherapy according to the method of Dupuy. *Harris.*

Preliminary Report Concerning the Passage of Bacteria Through the Tonsillar Tissue as Determined by Experimental Research.

DR. GEORGE B. WOOD, Philadelphia (*Transactions American Laryngological Association, 1913*): From a study of our experiments it seems that the following conclusions may be drawn: 1. The tonsils in the hog are more readily infected by the anthrax bacillus than any other portion of the buccal or pharyngeal mucosa. The clinical history of this disease in the hog shows that in the great majority of idiopathic cases the pharynx has been the site of invasion, and in all of these cases of pharyngeal diseases the tonsils are the port of entry. In none of my experiments was there any involvement of the pharyngeal or buccal mucosa other than the tonsils. While the culture of anthrax was generally brought into more intimate contact with one of the tonsils, it was impossible to limit the bacilli to the tonsillar surface, and they came into contact with a large part of the pharynx. In the infection an effort was made to rub the emulsion into one tonsil only and in one case the lesions were limited to one tonsil only, but this was not the tonsil on which the culture had been rubbed. 2. Anthrax bacilli penetrate through the cryptical and not the surface of the epithelium. 3. The anthrax bacillus probably gains access to the parenchyma of the tonsil by passing through the living, unaltered epithelium, and having gained access through the superficial layers of the epithelium, they tend to multiply in the deeper layers and then pass into the interfollicular tissue. 4. The anthrax bacilli penetrating through the living normal epithelium cause a devitalization of the tissue, which paves the way for secondary infection from the staphylococci or other pathogenic organisms. 5. The rapidity of the invasion is influenced both by virulence of the organism and the susceptibility of the individual animal. Following the invasion the subsequent course of the disease is similar to that found in other tissues. The toxin elaborated by the bacilli causes at first an accumulation of polymorphonuclear cells, later necrosis of the tissue cells with disintegration of the nuclei. The germinating follicles show more resistance to the disease than the interfollicular tissue. Associated with the necrotic process is an increase in the number and engorgement of the capillaries, and sometimes there is marked extravasation of the red blood

cells. The anthrax bacilli accumulate in the lymph spaces and also around the blood vessel walls. In some of the sections examined the bacilli were found penetrating the blood vessel walls, and a few were found actually in the blood current.

IV.—LARYNX.

Rheumatic Cricoarytenoid Arthritis.

U. CALAMIDE (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* May and June, 1912) reports two cases occurring in his own practice. In both the cricoarytenoid articulation was much swollen, reddened and completely immobile during phonation and respiration. The corresponding vocal cord was fixed in adduction, as is found in paralysis of the recurrent. There were evidences of rheumatism elsewhere in the body.

In the first case local and general treatment cleared up completely the laryngeal condition in the course of two weeks. In the second case, a more severe one with pronounced dysphagia, there was a disappearance of the tumefaction and pain, but at the end of two months there had been no change in the condition of the cricoarytenoid articulation and the corresponding vocal cord. *Harris.*

The Treatment of Dysphagia in Laryngeal Tuberculosis.

COMBIER (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* September and October, 1912). After referring to the various older methods employed, the author describes regional anesthesia of the trunk of the superior laryngeal as the most satisfactory means of relieving the difficulty. This can be secured either by the lateral route or the mediate route. The lateral route is the technic which he is wont to employ. A straight needle is employed, attached to a Pravaz syringe. The patient is placed on his back, the head slightly hyperextended, the neck is painted with tincture of iodin, a finger of the left hand which pushes the larynx toward the side to be injected, maps out the interthyrohyoid membrane. Frey advises introducing the needle midway between the superior cornu of the hyoid bone and the posterior superior angle of the thyroid cartilage. One goes in at this level always

a little nearer the hyoid bone, at first perpendicularly and then horizontally, from backward, forward, piercing the muscles which offer little resistance, and penetrating a little more than one cm. until we come in contact with the thyrohyoid membrane. At this point the patient should feel a pain in the ear if the nerve is reached. After being satisfied that blood does not follow the needle, the injection is used.

The writer makes use, as most others do, of one cm. of 85 per cent alcohol slightly warmed. The unpleasant results which sometimes follow the use of the alcohol can be avoided by preceding its use with half to one cm. of a weak solution of novocain and adrenalin. The method by the direct line, as recommended by Boncour, has the advantage of permitting a bilateral injection with the same introduction of the needle. There is a risk, however, of breaking the needle, and Combier feels that a second introduction is better. He states that it is rare that the injection is without effect; usually the results are excellent, and it is unattended by any unpleasant effects.

Harris.

Unilateral Affection of the Vocal Cords.

HEYMANN (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie*, January and February, 1913). The usual impression based upon textbook statements for many years is that a unilateral affection of the vocal cords is due to some specific condition. Heymann shows that this is not correct by quoting three cases that Rosenberg has reported showing this condition, which all recovered, and where tuberculosis, syphilis and cancer could be excluded. The fourth case was a case of Herbert Tilleys. Heymann himself has often had an opportunity of seeing isolated affections of one cord, or a part of one cord, due to chronic laryngitis, which persisted for a number of months, or even two years in one case, to finally get well as the result of treatment.

While this is true, he does not dispute the fact that the great majority of affections of the vocal cord have their origin in a constitutional disease. To explain this it is necessary to know exactly in regard to the etiologic conditions.

The exact genesis of catarrhal inflammation is not yet known, but the usual views held would tend to explain why in chronic laryngitis both cords are wont to be involved.

Harris.

The Early Diagnosis of Laryngeal Tuberculosis.

WILLIAM LAMBERSON (*New York Medical Journal,* October 19, 1912) has made an examination of the throats of 730 patients who have presented themselves at the tuberculosis clinics of the New York Department of Health. Of these, 244, or one-third, have been found to have tuberculous trouble in the lungs. Of these 244, 45 per cent have had tuberculous laryngitis, and 21 per cent had varying degrees of laryngitis, nontuberculous in origin, making a total of 66 per cent of positive chest cases with some pathologic laryngeal condition.

The author is of the opinion that in many of these cases of pulmonary tuberculosis the laryngeal condition, while apparently catarrhal, is really tubercular. He calls attention to the significance of a unilateral involvement. A cord showing loss of luster, rounded and thickened in parts, slow to respond in adduction, is strongly suggestive of tuberculosis. Later it assumes a bottle or fusiform shape, partly overlapped by a corresponding ventricular band.

The epiglottis in the early stages may show only a slight loss of mobility and change in appearance from the usual pink to a soft, velvety congestion. Later the color may be a deep red. As regards the voice, in his experience it is apt to be weak or husky, rather than hoarse. Incomplete adduction of one or both cords may be due to a lesion in the musculature or to pressure upon the recurrent laryngeal nerve. Pain is not a constant manifestation of tuberculosis of the larynx. Dysphagia is a symptom denoting the involvement of the deeper structures. He rightly recommends that all tuberculous patients should have early and frequent laryngeal examinations. In his experience the majority of cases start with hyperemia rather than anemia. *Harris.*

Thyrotomy for Cancer of the Larynx, With Report of Eleven Cases.

DR. D. CROSBY GREENE, Boston (*Transactions American Laryngological Association, 1913*): In a paper presented to this association in 1906 we reported the results of an investigation of the lymphatic drainage of the larynx by means of submucous injections of methylen blue and mercury. The results obtained are confirmatory of those reported by others in showing that the network of lymphatic vessels which extends beneath the mucous membrane throughout the interior

of the larynx is richer in the number and size of the vessels in the supraglottic region, relatively poorer in the subglottic portion, while on the vocal cords the vessels are very small and widely separated. These anatomic facts account for the slow growth and late development of the disease in the cervical lymph nodes in cases of epithelioma of the cords, and furnish an argument for the possibility of cure in early cases by the operation of thyrotomy and excision of the growth with a wide margin of healthy tissue. This is supported by the result of the operation in the hands of numerous operators, both in this country and abroad, so that at the present time it is almost universally recognized as the proper procedure for the treatment of early intrinsic cancer of the larynx. Certain details of the technic have an important bearing on the immediate and after-results of the operation. The steps of the operation are: 1. Ether by inhalation, preceded an hour before by ¼ grain of morphin and 1/150 grain of atropin. 2. With the head slightly extended a median incision is made, extending from the lower border of the hyoid bone to the lower border of the cricoid cartilage. This incision is carried down through the prethyroid muscles until the thyroid and cricoid cartilages and cricothyroid membrane have been definitely exposed. 3. A one per cent solution of cocain is injected through the cricothyroid membrane into the cavity of the larynx. 4. The patient is now placed in the Trendelenberg position and a thick pad placed under the shoulders to bring the larynx into prominence. 5. The cricothyroid membrane is next incised in the median line, and through this incision a swab of ten per cent solution of cocain is introduced and applied to the laryngeal mucous membrane. 6. The thyroid cartilage, after a pause of five minutes, is divided from below upwards. In young subjects this may be done with a knife, but in the majority of cases where the cartilage has become ossified, it is best to use strong curved scissors with dull points. 7. The thyroid wings are now widely retracted and an examination of the growth made under good illumination. 8. Beginning at the free margin of the thyroid cartilage on the affected side in front of the growth, the internal perichondrium is elevated from off the cartilage with a sharp elevator from before backwards to a line well behind the limits of the growth, as well as above and below it. All

the soft structures are thus freed from the underlying cartilage. 9. Parallel horizontal incisions are now made with scissors above and below the growth. These incisions are carried about one-half inch back of the posterior limit of the growth. 10. The growth with its surrounding tissue is now entirely removed with a wire snare by which the posterior attachments are severed. Much depends on the proper selection of cases. When the growth is so extensive, even though confined within the cavity of the larynx, that the larynx cannot be opened without cutting into the growth, recurrence is not only possible but probable.

Decannulation and Extubation After Tracheotomy and Intubation Respectively.

DR. CHEVALIER JACKSON, Pittsburg (*Transactions American Laryngological Association, 1913*): The different forms of laryngeal stenosis associated with difficult decannulation or extubation may be classified into the following types: 1. Pain. Breathing through the neck with a properly placed tracheotomy cannula is much easier than breathing through the mouth, that once the patient becomes accustomed to it for quite a while he does not feel that he is getting enough air through the mouth, even though the larynx is perfectly patulous. In addition to this there is a nervous cell habit arising from previous experience with the stenosis that terrorizes the patient the moment he feels the slightest dyspnea. 2. Spasmodic. This form of stenosis may be associated with panic, or may be excited by subglottic inflammation. It is usually overcome by the same means as those suggested for panic, together with the treatment of inflammatory conditions that may be present. Doubtless one of the chief causes of adductor spasm is the prolonged wearing of the intubation tube. 3. Paralysis. Bilateral ankylosis of the cricoarytenoid joints may prevent decannulation until the laryngeal stenosis is relieved. This operation is not to be advised except in such cases as have remained rigid for a period of twelve months or more, and this is not meant to include the fixation that is associated with malignant, tuberculous or luetic infiltrations. 5. Neoplasms. Decannulation in neoplastic cases will depend upon the nature of the growth and its curability. 6. Hyperplastic. 7. Cicatricial, (a) loss of cartilage; (b) loss of muscular tissue; (c) fibrous. The hyperplastic and

cicatricial types of organic stenosis preventing decannulation may be classified as follows: 1. Tuberculosis. 2. Lues. 3. Scleroma. 4. Acute infectious diseases, (a) diphtheria; (b) typhoid fever; (c) scarlatina; (d) measles; (e) whooping cough. 5. Decubitus, (a) cannular; (b) tubal. 6. Trauma, (a) tracheotomic; (b) intubational; (c) operative; (d) suicidal. Conditions outside of the paralytic and neoplastic forms are almost all the result of inflammation, often with ulceration and the secondary tissue changes. In the infective granulomata it is practically always the mixed infections from oral sepsis running that do the harm. The chief exception to this is diphtheria, which is in many cases a distinctly necrotic process. In the rare cases in which laryngeal tuberculosis of such severe type as to require tracheotomy is cured, decannulation presents little difficulty after the infiltrations are reduced. The reduction of these infiltrations by the galvanocautery through the laryngeal speculum is readly accomplished. Should cicatricial stenosis from ulceration remain, it is to be treated in the same way as cicatrices from other causes—by laryngostomy. In those old cases of luetic fibrosis little amenable to the older methods of treatment, salvarsan has accomplished wonders. Dr. Emil Mayer has recommended the use of radiotherapy in the treatment of scleroma. So far, however, the results have been so unsatisfactory that they practically constitute the only cases in which decannulation is impossible. When typhoid fever was prevalent in Pittsburgh, it was found that the ulcerative lesion in the larynx was practically always the result of mixed infection, but in some instances they were due to thrombosis of a small vessel with subsequent necrosis. The after-treatment of these cases is chiefly by prolonged intubation, and in some cases by laryngostomy. Scarlatina may be followed by acute laryngeal stenosis which is cicatricial. Occasionally foreign bodies may ulcerate through from the esophagus into the trachea. A properly fitting tube will not cause any ulceration, if it is free from roughness or sharp edges and is removed sufficiently often to be cleaned. For diphtheria and like conditions I have never seen any improvement on the original O'Dwyer apparatus. When a tracheotomized case reaches the stage when it is to be trained to breathe through the mouth, it is necessary to occlude the cannula. For the reduction of exu-

berant granulations, nothing has yielded better results than resorcin. As a stimulation to epithelialization the German preparation "scarlet red" (Biebrich) in a sterile 20 per cent mixture has yielded excellent results during laryngostomy. One of the most common causes is the neglect of frequent changes of dressings.

Primary Carcinoma of the Epiglottis, With Report of a Case.

DR. EMIL MAYER, New York, (*Transactions American Laryngological Association, 1913*): Primary cancer of the epiglottis is a rare disease, and up to the present time the treatment has consisted of surgical intervention or the use of radium and the X-ray. I have seen two cases of cancer of the epiglottis. The first case was diagnosed as probable malignant disease of the epiglottis, and the subsequent history was that of primary epithelioma of the larynx with subsequent metastasis, laryngectomy, and death. The second case was that of a man, 64 years of age, who consulted me for some slight difficulty in swallowing, especially the swallowing of cold liquids. His general condition was good. Examination showed some edema of the uvula, and numerous small white spits on the tongue and inner surface of the cheek. The condition was diagnosed as leukoplakia buccalis. He gave no history of lues. After treatment he improved somewhat, but this did not last, and some six or seven months later there was discomfort in swallowing, a nasopharyngeal catarrh, and the white patches still present. There were no other evidences of a diseased condition in the throat. About three weeks later a new condition presented itself on the laryngeal surface of the epiglottis. There was a deep ulceration surrounded by a thickened mass. The pathologist pronounced this cylindrical celled carcinoma. Here I had a case of carcinoma of the epiglottis in its earliest stages. The epiglottis was removed, after the patient had been anesthetized and the suspension laryngoscope placed in position. There was little bleeding at the time of the operation, but twenty-six hours after, the patient began to expectorate large quantities of blood. This bleeding was checked by the application of ice internally and by sprays of peroxid of hydrogen. He left the hospital in ten days. This was the first operation of its kind ever performed under the ingenious suspension laryngoscope of Killian.

V.—MISCELLANEOUS.

Diseases of the Eighth Nerve in Connection With Epidemic Parotitis.

MAUTHNER (*Archiv für Ohrenheilkunde*, Vol. 87, p. 223). The literature on this subject shows that permanent deafness in the affected ear is the result, temporary deafness the exception. A slight disturbance of the auditory nerve and the labyrinth have up to the present time not been described. While the most common period for involvement of the ear in connection with mumps is from the sixth to the fifteenth year, no thoroughly authenticated case has occurred over forty-one. The author makes the following divisions:

1. Cases with permanent abolition of function (deafness or deafness with loss of static irritability).

2. Marked disturbances: (a) With temporary abolition of function; (b) with temporary severe disturbance of function.

3. Slight disturbances for the most part only to be demonstrated by means of the continual tuning fork tests and exact examination of the vestibular apparatus.

1. Cases with permanent abolition of function. Here there is usually no involvement of the middle ear. At times a prodromal tinnitus occurs, usually on the third or fourth day of the disease, when the general symptoms begin to moderate, or during a new onset of fever with a secondary involvement of the glands near the ear. This may occasionally take place during convalescence as late as the fifteenth day. It is wont to be unilateral. The noise is of all degrees of severity from the slightest to the most intense. It is followed in the briefest interval of time by complete unilateral deafness. Bilateral deafness is also occasionally met with. In seventeen cases described by Fournies, nine were bilateral.

The characterization of apoplectic form seizure Mauthner does not approve of, inasmuch as the onset, while rapid, is by no means always sudden, there being cases on record where complete deafness has not resulted until at the end of five weeks. The disturbances of the vestibular apparatus can be of all degrees, from complete loss to entire preservation. The functional tests of two cases are given, illustrating the extremes. Vertigo is in the majority of cases associated from the beginning with the noises in the ear, separated only by

an interval of hours, and less often of days. It is wont to disappear in the course of several days. At times it can be of such an intensity as to lead to vomiting, and the patient is unable to go around. Slight degrees of vertigo will be apt not to be discovered.

2. Marked disturbances associated with either (a) temporary abolition of function, or (b) temporary severe involvements of function. The clinical picture of these cases in their onset and course does not differ from those in the first group. The author reports a case of complete loss of hearing which returned after eight days, and a second case where there was serious impairment, also restored in the course of eighteen days. Cases belonging to this group are not common.

3. Slight disturbances. For the most part only to be recognized by the accurate testing with tuning forks and the exact examination of the vestibular apparatus. The discovery of this group of cases is the result of the recent more accurate methods of testing the vestibular apparatus. The author recognizes the difficulty of finding such cases. In the course of two years he has had an opportunity of examining a hundred cases of parotitis, of different ages and both sexes. Of these one hundred cases, twenty-one were observed during the first four weeks of the attack of mumps. Of the hundred cases, in four, disturbances of the ear were shown which had a direct relation to the attack of mumps. The functional tests of these four cases are given in detail. The disturbances in question in three of the four cases had to do with the cochlear apparatus. In the fourth case, hearing for both the sound and the speech were involved. The impairment in all cases was found in the middle register between "c" and "a." With the exception of the fourth case, it was only slight, representing about 10 per cent, but repeated tests established its accuracy.

The author discusses at considerable length the views of Wittmaack in regard to a toxic involvement of the acoustic nerve, and reaches as the result of his studies the following conclusion as regards the pathology of the ear complication of mumps:

The clinical signs in all three groups resemble for the most part the picture of acute toxic and infectious neuritis of the eighth nerve. The slight disturbances (group 3), which are

here described for the first time, will tend to expand the limits of the clinical picture and cause us to suspect analogous disturbances of other acute injuries. *Harris.*

Poisoning From the Use of Menthol.

LEROUX (*Revue Hebdomadaire de Laryngologie, D'Otologie et de Rhinologie,* September 11, 1912) calls attention to the great danger of using menthol in concentrated form, or even in any strength, in the respiratory tracts of young infants. He reports a number of cases where asphyxia and almost death have occurred. This may be due to the (a) falling of the menthol oil in the larynx, (b) spasm of the glottis due to the contact of the oil in the larynx, (c) hypersecretion which is produced in the nasopharynx by the presence of the drug, (d) reflex irritation upon the lungs and heart as a result of the effect of the drug upon the nasomucosa.

He strongly urges that the drug should never be used in infants under three years of age, great care in children up to the age of seven, and in adults the employment of a dose not stronger than 10 cm. to 50 dr.

Apart from asphyxia, acute conjunctivitis, laryngeal cough, pseudoerysipelas of the face and noises in the ear have been met with as the result of the use of menthol, while in certain cases its continual employment produces an erythema of the lips and of the nose, and a condition akin to morphinism and cocainism, which may be termed mentholism. *Harris.*

The Diagnosis and Treatment of Cicatricial Stenosis of the Esophagus.

GUISEZ (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* Vols. XXXIV, XXXV, 1912) in 1200 esophageal examinations has met with 90 cases of cicatricial stenosis, making it, next to cancer, the most frequent condition met with in the esophagus. In addition to the usual causes for stenosis, he has met with cicatricial stenosis of inflammatory origin in a number of cases. Here there has been no wound or traumatism, but traces of subacute or chronic esophagitis which has little by little produced an inflammation and sclerosis of the walls. In the chronic form it is due to chronic irritations, most often alcohol, or the esophagus may be incompletely compressed by a growth from without. Most fre-

quent of all the causes, however, is spasm, usually the result of improper mastication.

Guisez lays much importance upon the previous history. Among physical signs he speaks of inspection, palpation and auscultation. Catheterization is condemned. The three modern methods are radioscopy, radiography and especially esophagoscopy. Catheterization with a soft bougie he advises to precede the endoscopy, in order to determine the probable situation of the stenosis. He is in the habit of making the examination in the Rose position, and finds local anesthesia sufficient except in children.

Where the stenosis is due to a foreign body, simple ulceration or an esophagitis, it is usually single. Stenosis due to caustics is almost always multiple. The usual site of the stenosis is either at the beginning of the esophagus or at the cardiac end. Stenosis of the cardia is most frequent of all, 33 to 40 per cent. In the diagnosis of the esophageal stenosis, spastic contractions are to be borne in mind. Occasionally also congenital stenosis will be met with. While the history is of value, it sometimes is misleading. Patients will give a definite cause to the stenosis when the condition may be of a malignant nature, for example. As complications he mentions obstruction from the bolus of bismuth which had been swallowed in connection with the radioscopic examination. The inflammation of the mucosa can go on to a phlegmonous condition, localized or diffuse. Where the mediastinum is involved, death is wont to occur very rapidly. The prognosis of cicatricial stenosis is bad. Billroth in a series of cases from 1877 to 1886 reported a mortality of 38 per cent. It may be stated that abandoned to itself, a cicatricial stenosis is fatal sooner or later. The prognosis depends to a considerable degree upon the age of the patient.

The treatment has two objects in view: the dilatation of the stricture sufficient to permit nourishment being swallowed, and second, to effect alterations in the cicatricial tissue of the walls of the esophagus. Dilatation by means of the esophagoscope is wont to produce much better results than blind dilatation. Where the stricture yields easily, it is possible to rapidly increase up to No. 18 or 20 at the first sitting. In cases where it is more dense, it cannot be possible to use the bougie higher than No. 13. In adults, 20 mm., in children

under six years, 6 mm., and from six to fifteen, 16 mm. should be the maximum of dilatation.

In two particularly difficult cases, Guisez has succeeded by means of the Plumer thread. In certain cases he has employed with advantage the Laminaria dilatation as recommended by Ebstein; also, that of v. Acker, which consists in a drain, by means of a stiff bougie left in position. His best results have been obtained, however, by dilator drains of rubber or metal in the shape of a collar button and retained in position by means of a silk thread fastened at the upper end. Esophageal dilatation should not be undertaken for a number of weeks after the burn, or until all the acute inflammation has subsided. When the dilatation is easy, the introduction of the bougie with the esophagoscope can be abandoned in favor of simple dilatation. The patient should not be allowed to dilate his own strictures until it is certain that the introduction of the bougie can do no harm. The author has used internal esophagotomy for many years with success and has a special instrument for that purpose. More recently, however, he has abandoned his procedure in favor of electrolysis, for which he has devised a set of electrodes with which he has had excellent results. *Harris.*

Lactic Ferment in Rhinology.

Sune y Medan (*Archives Internationales de Laryngologie, D'Otologie et de Rhinologie,* January and February, 1913) has employed the lactic ferment, either in the form of powder or solution, in various affections of the nose. In acute rhinitis the attack can be decidedly shortened by the use of this remedy. He is in the habit here of making use of the powder, introduced, if necessary, after the turbinals have been shrunkened with a weak solution of cocain. The canula should be carried high up into the nose. The insufflation will have to be repeated six to eight times during the day. The results are uniformly good.

In catarrh of the nasopharynx the pulverized ferment is applied through the mouth, the canula being carried in behind the palate. In certain cases, in addition, the nose is to be painted with the liquid lactovacilline and immediately afterwards the powdered ferment insufflated into the nose. The results here are also good. The viscosity and the bad odor

are wont to disappear and the patients find themselves much relieved.

In ozena he pursues the following technic: Cleansing of the nose, removing of the crusts, if necessary; second, the large insufflation of the lactovacilline powder; in certain cases, in place of the powder, liquid vacilline has been used. The patient returns every week or every two weeks for observation. He states that he can truthfully say he has never seen a case of atrophic rhinitis which has not been more or less benefited by the treatment. The advantages of the treatment are its ease of application and the absence of all irritating effects. The patient is able to make the applications at home. It is possible in 40 per cent of the cases to show that a cure has taken place in from four to eight months. By cure is meant a disappearance of the crusts and of the odor.

Harris.

Further Observations on Some Anatomic and Clinical Relations of the Sphenoid Sinus to the Cavernous Sinus and the Third, Fourth, Fifth, Sixth and Vidian Nerves.

DR. GREENFIELD SLUDER, St. Louis (*Transactions American Laryngological Association, 1913*): In a previous communication (last May), in which the question was raised as to the etiology and treatment of migrain, Sluder made the statement that he believed that many (but not all) of the recurrent headaches which bear the name of migrain are sphenoidal empyemata which have lost most, if not all, local signs, or which were started as such empyemata, and that the nerve trunks had become involved either by extension of the inflammation (or its toxin) through the thin wall separating the sphenoid sinus from the adjacent nerve trunks. The results obtained during the past year in a goodly number of cases strengthen his belief in the correctness of the deductions detailed in that report.

Sluder observed that the third, fourth, and three divisions of the fifth, sixth and Vidian frequently lie in close association to the sphenoid sinus, a deduction drawn from specimens which he studied by cross sections. His findings, with the exception of the Vidian nerve, were corroborated by Ladislaus Onodi (*Archiv. fuer Laryngologie*, Bd. VI, Heft II, July 10, 1912). This author's method was to follow the nerve trunks in certain specimens, sometimes to remove the wall of

the sphenoidal sinus, and then to study the relations of the nerve trunks thereby exposed. He found that they were in these close associations for varying distances, sometimes even as much as 20 mm.

He did not consider the cavernous sinus in these relationships. He pictures specimens where the sphenoid sinus extended so close to the clivus of Blumenbach as to make transparency of the separating bone, and shows how this brings the sixth into these associations. From an inspection of Dr. Warren B. Davis' 145 Caucasian specimens showing the nose and accessory sinuses from the eighth week of fetal life to the twenty-fifth year uninterrupted (several specimens for each year, except the eleventh year), Sluder observed that the sphenoid sinus spreads laterally at an early age, reaching to close proximity to the second division of the fifth as early as two and one-half years, and that this condition runs almost constant throughout the series. Its development (Davis) begins in the anterolateral aspect of the body, and slowly extends backward, spreading, however, rapidly laterally to approach the foramen rotundum, and then proceeding backward. As early as the sixth year the Vidian canal may be approached. Sluder considers that if he is right in the conclusion that the mode of production of these headaches—the pathologic sequence—is the close association of the sphenoid sinus to the nerve trunks, and that the inflammatory processes are transmitted through the thin bone separating the cavity of the sphenoid from the associated nerve trunks, then it is necessary that such anatomic associations be formed in early life as an explanation of such headaches beginning in early life. Another year of clinical observation strengthens Sluder's belief that the pathologic process underlying these cases is a hyperplastic sphenoiditis. From an observation of 100 cases he concludes that the second division of the fifth and Vidian are the nerves most frequently involved (95 per cent). They may be involved singly, or together, then making the picture which would otherwise emanate from the sphenopalatine ganglion. It is difficult to differentiate this class of cases from sphenopalatine ganglion neuralgia; hence, one should be carefully on guard. Since the previous communication, three medicines have been tried for intrasphenoidal use: Iodid of potassium in water, 2 to 5 per cent, proved to pos-

sess nothing upon which its use may be recommended; it is apparently inert. One per cent chlorotone in water proved to lack recommendations. The proprietary "cresatin" also failed to prove better adapted for these purposes than carbolic acid or oil of wintergreen. The medicines which have so far proved of greatest benefit are: One per cent carbolic acid in oil; 2 to 10 per cent oil of wintergreen and aqueous solutions of sodium salicylate, 2 to 5 per cent. These have been successful in allaying the pain long after the sinus was satisfactorily opened and the wound healed.

Foreign Bodies in the Esophagus, With Report of Two Cases.

Dr. Cornelius G. Coakley, New York (*Transactions American Laryngological Association, 1913*): The first patient, a boy 16 years of age, thought he had swallowed a piece of plate in his soup. He had a temperature of 105,° rapid pulse and pneumonic area in the right lung, when first seen. Examination of the pharynx showed edema extending from the vault of the nasopharynx as far down as one could see with the laryngeal mirror, or feel with the fingers. It much resembled a retropharyngeal abscess. The X-ray showed a triangular shaped foreign body. It was jagged and evidently had cut into the mucous membrane so as rapidly to infect the pharyngeal mucosa, and the secretion from the infected pharynx passing into the larynx, setting up a septic pneumonia within twenty-four hours. The boy died fourteen hours after the operation, from acute septic pneumonia. This case shows the necessity for the prompt operative relief for removal of sharp foreign bodies. The same day I saw a boy three years of age who had swallowed a coin. The X-ray showed the coin at the level of the sixth cervical vertebra. The child had no symptoms. The coin was removed by operation and the child suffered no subsequent discomfort. In this instance the coin had been in position for five days before it was removed.

Phlegmons of the Upper Respiratory Tract.

Dr. John O. Roe, Rochester (*Transactions American Laryngological Association*, 1913): The phlegmon is simply another name for inflammation, although clinically it is regarded as an extraordinarily intense inflammation and looked

upon as being of more or less malignant character. We may have an inflammation of this character in any part of the body, the intensity of which may depend upon the virulence of the infection and the resistance or lack of resistance of the patient, or of the tissues, to such infection. Owing to the difficult breathing or choking which such inflammation causes in the upper air passages, it has received the name of angina. Inflammations of a severe grade, termed phlegmons, may involve any portion of the upper respiratory tract. We almost invariably find a phlegmon attacking one whose general condition has become weakened. The treatment of these infections requires no special law unto itself, and must be dealt with according to the location and associated complications.

Nitrous Oxid Gas, Essence of Orange, Ether and Sequestration in General Anesthesia for Operation in the Upright Position.

DR. THOMAS R. FRENCH, Brooklyn (*Transactions American Laryngological Association, 1913*): We are now able to state that as a result of the movements of the new operating table, and also as a result of the tests made in our clinic at the Long Island Hospital, that operative work in the upright position can be done with still less loss of blood, with the need of still less of the anesthetic, and with less disagreeable conditions during recovery from the anesthetic. Sudden changes in posture or sudden disturbances of the body while the patient is under a general anesthetic, predisposes to shock, the effects of which are manifested both during the operation and during the recovery from anesthesia. After a careful study of this phenomenon, I became impressed with the value of elevation of the body from the recumbent position to the upright position without jarring, and this led to the construction of the chair table. By the aid of a new attachment all the movements of the body are now made with as great freedom from jarring and disturbance of balance, with infants and very small children, as had hitherto been possible only with adults or larger children. The resulting improvement in conditions leaves no doubt of the accuracy of the observation. One of the most important and valuable recent additions to methods of anesthesia is the ability to omit or bridge the second stage, or stage of excitement, and we have been impressed with the desirability of attaining narcosis

without struggle. This can be accomplished with nitrous oxid gas, but with greater ease and certainty with the essence of orange. The discoverer of the remarkable effects of the essence of orange as a preliminary to ether, Dr. Gwathmey, of New York, has demonstrated the possibilities and has proven to our satisfaction that it greatly assists in the reduction of shock by bridging the stage of excitement. Anesthesia conducted in this way up to the time of raising the body to the upright position is a contributory factor in reducing hemorrhage and also in reducing the quantity of anesthetic and in shortening and modifying the anesthetic after-effects. Irrespective of all other conditions, there is a well-defined relationship between the degree of skill in which a patient is anesthetized in the upright position and the amount of hemorrhage is reduced if the anesthetic is smoothly given. A preliminary use of nitrous oxid gas or the essence of orange and the drop method of anesthol, followed by the drop method of ether with the open inhaler, and later with the Allis inhaler, will insure a prolongation of the anesthesia after the body has reached the upright position, a reduction of hemorrhage, and a reduction in disagreeable sequelæ. Sequestration of the limbs produces a congestive hyperemia, and is produced by means of inflated blood pressure cuffs about the thighs and arms; this reduces the amount of blood in the head. This, in association with the upright position which has been carried out in fifty-eight cases during the past season, reduces still further the amount of anesthetic required and still further the loss of blood. There need be no apprehension of subsequent bleeding.

History of a Broncholith, Bronchial Calculus, or Lung Stone.

DR. WALTER F. CHAPPELL, New York, (*Transactions American Laryngological Association, 1913*): This case is cited to show that all causes of asthma are not centered in the nose, nor all throat coughs in the larynx. The patient was a woman, 52 years of age, who was attacked by severe wheezing, the spasmodic cough lasting an hour at a time, and by intense tickling in the throat. The tickling was below the larynx, and the wheezing in the upper part of the chest, in front and behind. The patient soon raised a small calcareous mass, but the asthma continued. Another coughing spell resulted in the expulsion

of a large calcareous mass. Immediately all wheezing stopped and the breathing became absolutely free. Later another calcareous mass was coughed up. Chemically these masses consisted of phosphate of lime. The patient gave no history that would give a clue to the cause for the existence of this condition.

The Removal From the Esophagus of a Plate of False Teeth Embedded for Eighteen Years, by Means of the Esophagoscope.

DR. D. BRADEN KYLE, Philadelphia (*Transactions American Laryngological Association, 1913*): This foreign body which I present was very difficult to locate, in spite of its size and shape, on account of the granulation tissue which had organized into fibrous tissue, together with the curvature of the spine, as the X-ray picture shows. The patient swallowed the upper suction plate with four front teeth attached while sleeping. The plate lodged in his throat, and a physician who was called in wished to push it down into the stomach, to which the patient objected. He then went to a hospital where, after the passage of several instruments, he was assured that he had never swallowed the plate or that it was no longer in the esophagus. For a few weeks after the patient felt slight pain in the lower part of the neck at the point he always felt that the object had lodged. After that time there was no sensation, but swallowing has always been difficult. The Kahler esophagoscope was used, and many attempts were made before this foreign body was successfully removed. By loosening the plate from the fibrous bed by setting up slight inflammatory action and then waiting a few days, the plate was removed without much ulceration of the structure.